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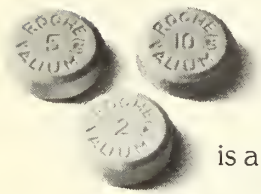


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A character all its own.



Valium (diazepam/Roche) is a benzodiazepine with a character all its own. Pharmacologically, it is a potent skeletal muscle relaxant and anticonvulsant (in adjunctive use), as well as an antianxiety agent. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

Valium® (IV)
diazepam/Roche

2-mg, 5-mg, 10-mg scored tablets

a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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The Kansas Press Looks at Medicine

Reluctant Advertisers

The proposed rules which would regulate medical advertising in Kansas leave no doubt about one issue. The advertising idea is not especially popular in Kansas.

Medical leaders said that rather plainly to an interim committee of the Legislature, which has been looking into the issue. All recognized, however, that the ground rules have been changed. Strict prohibition against advertising amounts to a violation of free speech by those who may want to advertise.

And so, with rules proposed this week by the Legislative committee, commercial advertising by Kansas physicians, osteopaths and chiropractors would be regulated by the state.

While printed ads could cite the name of the doctor, as well as the specialty, background training and certain credit data, there could be little else. No drawings, illustrations, animations, or dramatizations.

Those restrictions illustrate, without drawings or animations, the lack of enthusiasm for the idea.

The value in expanded advertising lies in helping educate and inform the great numbers outside the medical profession who invest so much in that business these days. Pictures, drawings, animations help communicate ideas, improvements . . . all sorts of things.

When the use of these most advanced communications aids is denied in that educational process, the value of the technique is reduced.

It is as if the medical profession decided that CAT scanners should not be used because they are so modern. — *Hutchinson News,* Oct. 21, 1979.

When Doctors Advertise

Standards have been proposed to control medical advertising in Kansas. It's a good idea. The Legislature's interim Committee on the Judiciary has proposed a list of advertising "dos and don'ts" to forestall the advent of any false, misleading or demeaning advertising practices.

The battle to permit commercial advertising by doctors and attorneys was an emotional confrontation, which finally was resolved in the courts. In separate decisions, it was ruled that the constitutional mandate of free speech entitles physicians and lawyers to advertise if they want to.

Still, the serious responsibilities that go along with these two highly important professions demands that the highest standards of fairness and good taste be used in presenting commercial advertising to the public. This feeling was underscored when the Kansas Board of Healing Arts and the Kansas Medical Society went on record during the committee hearings as still opposing the concept.

The Kansas Supreme Court sent down a set of standards for lawyers in February, and the Legislature will have a chance to enact similar guidelines for doctors in the upcoming session.

The proposed set of standards would regulate commercial advertising for physicians, osteopaths and chiropractors practicing within the state. It permits name, field of practice, schools attended, fees and whether credit cards are accepted to be included in medical ads. Forbidden are drawings, animations, illustrations, dramatizations, slogans, music, lyrics or testimonials.

It has become a difficult task in this ever-

(Continued on page 17)

ExTOL™



The Burdick Exercise Tolerance System helps track down coronary disorders.

Introducing Burdick's ExTOL the coordinated Exercise Tolerance System. Now from one source, the instrumentation components necessary to better detect clinically unsuspected or "silent" coronary disorders. All built, and backed, by Burdick, with the same quality that has made Burdick a leader in medical instrumentation for more than 65 years. The ExTOL System includes:

Burdick's TMS-300 Treadmill. A treadmill that lets you start your patient testing sequence at zero miles per hour; an important contribution to patient safety, especially the older patient. Infinitely variable speed control 0-7 miles per hour. Elevation to a maximum of 25% grade. Wide walking belt area for an extra margin of safety. Durable, curl-resistant heavy polyester belt tracks well. Remote control unit with digital timer and ECG start/stop control for automatic-start ECG instruments. Speed-setting caution indicator for added safety.

EK-6 3-Channel Electrocardiograph. Complete 12-lead ECGs i 10 seconds. Automatic lead marking. Lead lengths, selectio standardizations, sensitivity settings, paper speed, and lead chec can be automatically controlled. Heated styli and new BlueTrac paper produces clear, accurate, smudge-free ECG tracings.

CS-625 Monitor/Heart Rate Meter. Viewing screen project non-fade ECG display with freeze frame capability. Clear, easy-t read, digital heart rate meter with range from 12-240 beats pe minute. Heart-rate-limit alarm available.

DC-190 Defibrillator. Designed with the patient's an operator's safety in mind, simple operation. Energy levels fro 5-400 joules in easy-to-select detented positions.

Install ExTOL, Burdick's Exercise Tolerance System. If yo want accurate test results and control of the test sequence, yo want Burdick. ExTOL components also available separately if yo want to update your present system.

For a demonstration or more information, call **816-231-1900.**

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Tagamet®

brand of

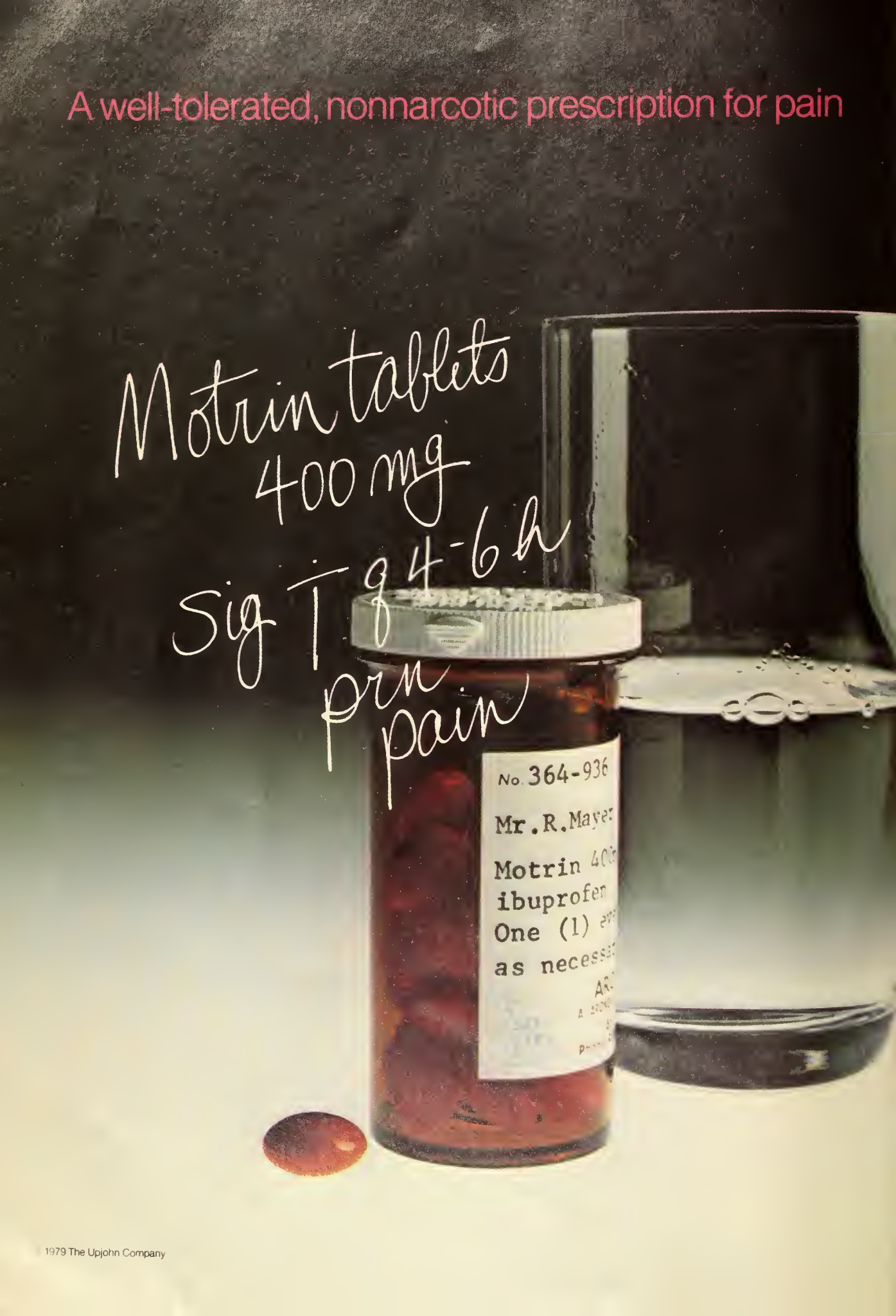
cimetidine

How Supplied:

Pale green 300 mg. tablets
in bottles of 100 and Single Unit Packages of 100
(intended for institutional use only).
Injection, 300 mg./2 ml.,
in single-dose vials
and in 8 ml. multiple-dose vials,
both in packages of 10.

SK&F LAB CO.
a SmithKline company

A well-tolerated, nonnarcotic prescription for pain



1979 The Upjohn Company

Motrin® now proved an effective analgesic for mild to moderate pain

Motrin 400 mg provided greater relief of pain than did propoxyphene 65 mg in controlled clinical pain studies.

Time after drug administration (hour)		.5	1	2	3	4
Mean relief-of-pain scores* (No. patients reporting)	Motrin 400 mg ibuprofen	.89 (108)	1.25 (108)	1.36 (108)	1.28 (107)	1.19 (106)
	Darvon 65 mg propoxyphene	.66 (100)	.99 (99)	1.13 (96)	.99 (96)	.80 (96)
Statistical significance		$p<0.02$	$p<0.01$	$p<0.05$	$p<0.02$	$p<0.002$

*0 = No relief 1 = Partial relief 2 = Complete relief

Data on file at The Upjohn Company

Motrin demonstrated statistically significant greater relief of pain than did Darvon at all time intervals.

Motrin 400 mg TABLETS
ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming
- Rapid analgesic action • Indicated in acute and chronic pain
- Well tolerated. The most common side effect with Motrin is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen)
now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea;* epigastric pain;* heartburn;* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness;* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.
Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

Caution: Federal law prohibits dispensing without prescription.

For additional product information, see your Upjohn representative or consult the package insert.

MED B-4-S

Upjohn THE UPJOHN COMPANY
Kalamazoo, Michigan 49001 USA

Remember

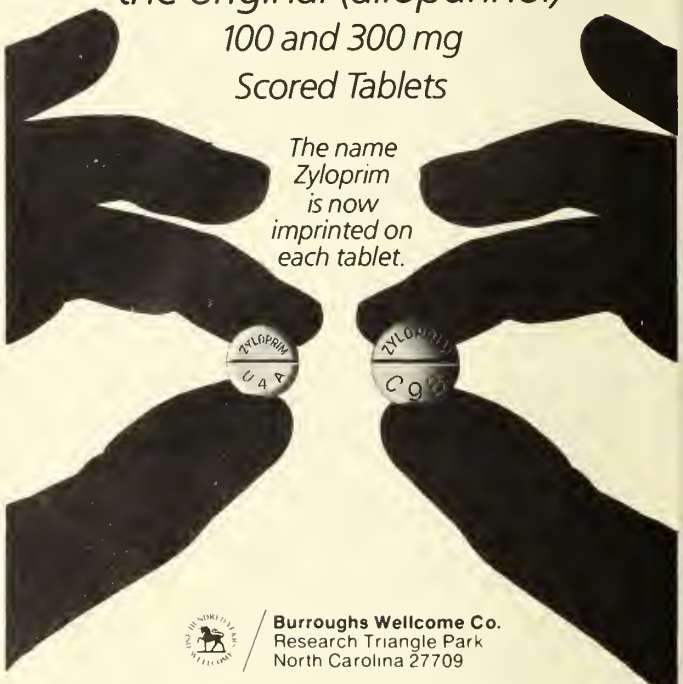
ZYLOPRIM®

the original (allopurinol)

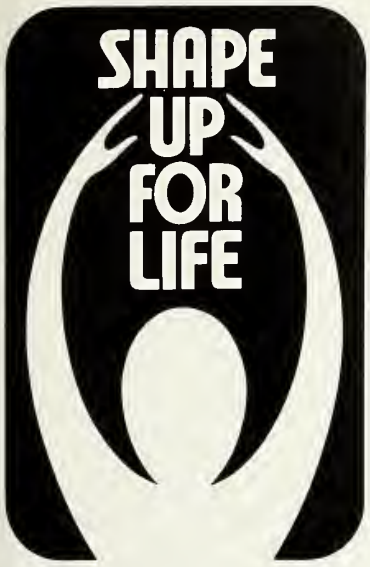
100 and 300 mg

Scored Tablets

The name Zyloprim is now imprinted on each tablet.



Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709



American Medical Assaciation Auxiliary
535 No. Dearborn St., Chicaga, IL 60610

Auxiliary News

The annual Fall Conference was held on November 27 and 28 at the Salina Hilton Inn. An in-depth communications seminar was conducted by Phil Coleman of Salina. Mr. Coleman has been an instructor of self-improvement classes for the past nine years at the Brown Mackie College, and conducts many workshops throughout the state. With combined humor and serious moments, Phil led us through a three-hour class on positive thinking, motivation, assertiveness — that is, taking charge of your life — leadership, transactional analysis, and time management. The seminar was planned to assist interested Auxilians in becoming leaders in their communities. All who attended felt it was very worthwhile and stimulating.

The Board of Directors met on the following day for a business meeting. The health projects chairman reported that the Learning Center on Body Pollution is gaining momentum across the state, as five more counties will be or are in the process of building their own units. To know that the school administrators and teachers are as excited as we are about this program is indeed heartening. Peggy Grillot, the AMAERF chairman, reported that mid-year contributions total $5,271.86. Our next meeting will be at the annual convention in May, in Kansas City.

In the following months we will continue to visit county auxiliaries and prepare for the annual convention. We hope to meet your spouse on one of these visits. Doctor, please help us. Even if you don't have time to be an active Association member, see that your spouse joins us. Our members are well informed and often are the ones who bring home information pertinent to your practice of medicine.

And if you are an involved KMS member, become even more committed by enrolling your spouse in the Auxiliary. Often all it takes is a request from you that she/he join us. You can even make it easier by sending in that check to cover county, state, and national dues.

Sometimes the old cliches persist, and the Auxiliary may seem to be just another social organization. It isn't! It is an organization that shares your concerns for the provision of quality health care. And we can help you. Our Auxiliary members are actively engaged in furthering legislation that will help you practice the kind of medicine you want.

Help us to help you. . . .

Sincerely,
Kathy Wedel
President
Kansas Medical Society Auxiliary

For the treatment of alcoholism —

HYLAND CENTER

- **A New Start**
- **A New Hope**
- **A New Way of Life**

10020 Kennerly Road
St. Louis, Missouri 63128
(314) 525-7200

A Division of St. Anthony's Medical Center

Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability insomnia associated with anxiety neuroses and transient situational disturbances; anx associated with depressive symptoms and as a treatment of symptoms of anxiety if such sy toms are a significant feature of functional or organic disorders, particularly gastrointestina cardiovascular

Effectiveness in long-ferm use, i e , more than 4 months, has not been assessed by syst atic clinical studies Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucom

Warnings: Not recommended in primary depressive disorders or psychoses. As with all C acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, an diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with ba furates and alcohol have occurred following abrupt discontinuance of benzodiazepi (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating) Ad tion-prone individuals, e g drug addicts and alcoholics, should be under careful surveilla when on benzodiazepines because of their predisposition to habituation and dependen Withdrawal symptoms have also been reported following abrupt discontinuance of benz azepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid o sedation

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may resu symptoms like those being treated anxiety, agitation, irritability, tension, insomnia and oc sional convulsions

Observe usual precautions with impaired renal or hepatic function

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazep has not been shown of significant benefit in treating gastrointestinal or cardiovascular com nent

Esophageal dilation occurred in rats treated with lorazepam for more than 1 yea 6mg kg day No effect dose was 1 25mg/kg/day (approximately 6 times the maximum hur therapeutic dose of 10mg/day) Effect was reversible only when treatment was withdrawn wi 2 months of first observation Clinical significance is unknown, but use of lorazepam for longed periods and in geriatric patients requires caution and frequent monitoring for sympto of upper G I disease

Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia, some have elevations of LDH As with other benzodiazepines, periodic blood counts and liver function t are recommended during long-term therapy

CLINICALLY SIGNIFICANT DRUG INTERACTIONS Benzodiazepines produce CNS depres effects when administered with such medications as barbiturates or alcohol

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerge rats during an 18-month study No studies regarding mutagenesis have been performed.

PREGNANCY Reproductive studies were performed in mice, rats, and 2 strains of rabb Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastrosch malformed skull and microphthalmia) were seen in drug-treated rabbits without relationshi dosage Although all these anomalies were not present in the concurrent control group, t have been reported to occur randomly in historical controls At 40mg/kg and higher, there evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lo doses Clinical significance of these findings is not known However, increased risk of cong tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam meprobamate) during first trimester of pregnancy has been suggested in several stud Because use of these drugs is rarely a matter of urgency, use of lorazepam during this per should almost always be avoided. Possibility that a woman of child-bearing potential may pregnant at institution of therapy should be considered Advise patients if they become pr nant to communicate with their physician about desirability of discontinuing the drug

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazep and its glucuronide

NURSING MOTHERS It is not known if oral lorazepam is excreted in human milk like ot benzodiazepines As a general rule, nursing should not be undertaken while on a drug sin many drugs are excreted in milk

Adverse Reactions, if they occur, are usually observed at beginning of therapy and gener disappear on continued medication or on decreasing dose In a sample of about 3,500 anxi patients, most frequent adverse reaction is sedation (15 9%), followed by dizziness (6.9 weakness (4 2%) and unsteadiness (3 4%). Less frequent are disorientation, depression, n sea change in appetite headache, sleep disturbance, agitation, dermatological symptoms, function disturbance, various gastrointestinal symptoms and autonomic manifestations. In dence of sedation and unsteadiness increased with age. Small decreases in blood press have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind that multiple age may have been taken Manifestations of overdosage include somnolence, confusion and coi Induce vomiting and/or undertake gastric lavage followed by general supportive care, monit ing of vital signs and close observation Hypotension, though unlikely, usually may be control with Levarterenol Bitartrate Injection U S P Usefulness of dialysis has not been determined

Ativan® (IV) for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase do gradually when needed, giving higher evening dose before increasi daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosa may vary from 1 to 10mg/day in divided doses. For elderly or deb tated, initially 1-2mg/day; insomnia due to anxiety or transient situ tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories
Philadelphia, PA 19101

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Why one benzodiazepine and not another?

Are you concerned about long-acting metabolites? Many clinicians, as well as pharmacologists, are beginning to draw attention to this problem (see New England Journal of Medicine, April 5, 1979).

In contrast to some older benzodiazepines, Ativan (lorazepam) does not give rise to long-lasting active metabolites. As with all benzodiazepines, you should follow the usual precautions concerning co-administration with other CNS depressants and warn your patients against operating dangerous machinery and motor vehicles.

However, it is noteworthy that Ativan showed no clinical evidence of accumulation even when given in high doses over periods up to 6 months. The half-life of free lorazepam is about 12 hours; steady-state serum levels are attained in 2-3 days. Comparable data for diazepam: 20-50 hours and at least 7-10 days. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Ativan has a convenient b.i.d. or t.i.d. dosage schedule; it is compatible with a long list of other medications and, of course, it is a highly effective anxiolytic agent, as established in numerous nationwide, double-blind, controlled evaluations in thousands of patients.



See important information on preceding page.

Ativan® (lorazepam) for Anxiety

V-Cillin K®

penicillin V potassium

is the most widely prescribed brand of oral penicillin



Tablets
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VITAMIN C AGAINST CANCER, by H. L. Newbold, M.D. Stein & Day, New York, 1979. 363 pages. $10.98.

Dr. Newbold's book arrived with a separate publisher's news release, decrying the just published results of a controlled Mayo Clinic study of vitamin C in cancer patients. That the double-blind controlled study done at the Mayo Clinic failed to demonstrate any survival advantage for the vitamin C treated patients must have created a temporary commercial apoplexy at Stein and Day. Your copy of Dr. Newbold's book — if you decide to order one — will also contain a copy of the publisher's disclaimer of the Mayo study. However, you will need to find your own copy of the negative Mayo study which was published in the September 27, 1979 issue of the *New England Journal of Medicine.*

Vitamin C Against Cancer is an admixture of interviews with scientists who either discovered vitamin C (Szent-Györgi), or who see vitamin C as a panacea for disease; along with these illustrious names are physicians whose anecdotal personal experience has convinced them of vitamin C's efficacy in the treatment of patients with "terminal" cancer. The author belongs to this latter group, editorializing throughout the text, and in a series of chapters detailing the specifics of how he approaches cancer patients nutritionally, with the expected emphasis on details of vitamin C administration. Dr. Newbold correctly identifies the failure of more established clinical research groups to stress the importance of nutritional support of patients being treated for advanced malignancies. Unfortunately, the author fails to carefully define "terminal" patients who — all too often in the minds of lay persons and physicians alike — achieve "terminal" status the moment a diagnosis of recurrent cancer is established.

Disparagement of the medical establishment and the government's financial support of cancer research will undoubtedly improve the commercial appeal of Dr. Newbold's book, but will accomplish little in identifying the proper role of vitamin C in cancer treatment. The author is extremely proud of his maverick status, and in the design of his book gives equal time to another maverick, Dr. Bross, a statistician with cancer data evaluation experience, which allows him to make the following fatuous statement, "If I was advised to take radiation I would know at that point I was terminal. I wouldn't bother." The foolishness of such inexpert testimony is perpetuated by the author's own inane comments, "The academic people haven't been in the bullpen, so to speak" (p. 191).

Such nonsense shouldn't invalidate the worthwhile chapters, where the author interviews Drs. Burchenal and Good. Both interviews are informative and are fair representatives of current investigative endeavors, plus state-of-the-art chemotherapeutic generalizations. However, one must be cautious in reading the author's own interpretations of appropriate treatment for oat cell lung cancer and testicular cancer, as his simplistic generalizations in these areas are inaccurate.

The "scientific" underpinning of the vitamin C story deserves a close look. The complete study of Cameron and Pauling can be found in Appendix A of Dr. Newbold's book, a study that compares 100 "terminal" cancer patients treated with vitamin C with 1000 untreated controls. Use of 1000 historic controls serves to dilute mean survival in resistant cancers (colon and lung), providing the relatively small numbers of vitamin C treated patients (13 colon and 15 lung) an inherent survival benefit. That Drs. Cameron, Pauling, and Newbold chose to ignore the placebo effect of vitamin C treatment (plus the possible hydration benefits of intravenous fluids) is totally ignored. But one shouldn't be surprised by the "statistically significant" accomplishments of "scientists" who pride themselves in ignoring the

(Continued on page 24)

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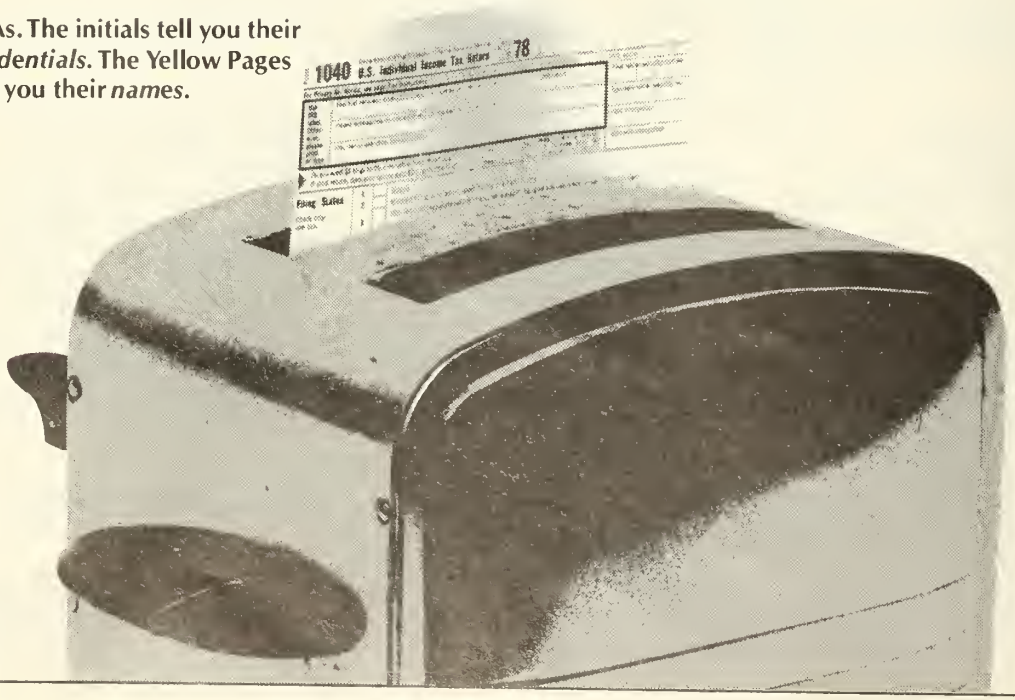
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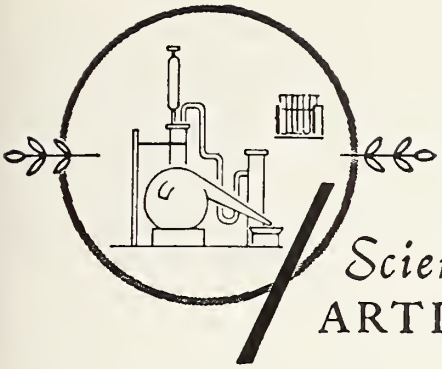
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Auditory Evoked Potential

Clinical Applications of Brainstem Electric Response Audiometry

RICHARD J. CUMMINGS, M.D. *and* **THOMAS L. SCHRODER, M.A.**, *Wichita*

Brainstem electric response audiometry (BERA) is an effective diagnostic tool for both otologists and audiologists. Its utilization provides the means for confident diagnosis of some difficult or impossible cases.

RESPONSE TO SOUND stimuli of the human electroencephalogram has been known for many years. A great deal of research has been devoted to making these responses applicable to the evaluation of human hearing. Practical use of these responses was made possible in 1954 by the development of electronic averaging which allowed these responses to be differentiated from other ongoing EEG activity.[1]

By the early 1970s, the equipment used for brainstem electric response audiometry (BERA) was perfected to the point where it became a useful clinical tool in otology. BERA is the measurement and recording of electrical potentials as they occur along the eighth cranial nerve and ascending auditory pathways in the brainstem as a result of specific auditory stimulation. This technique is valuable in the differential diagnosis of cochlear, retrocochlear, and functional hearing losses. It is especially valuable in that it does not require the active participation of the patient and can even be performed under general anesthesia.[2]

Technique

For clinical assessment of the peripheral hearing mechanism, a series of rapid stimuli — usually clicks, tone bursts, or logons — are presented through earphones or loudspeakers; electrical potentials arising from auditory stimulation are detected as far field responses through surface electrodes attached to the vertex and to each mastoid. These electrical potentials are amplified and sent to a computer which averages the electric response. Because the response is time-locked relative to the auditory stimulus, it can be extracted from other ongoing random activity. The averaged electric response to a series of auditory stimuli is converted to a graphic representation, such as on an oscilloscope or strip chart, and displayed as wave forms.

In the first ten msec following auditory stimulation, at least five waves can be identified *(Figure 1)*.[3] Each wave represents the averaged electrical potentials generated at locations along the auditory pathway. It is generally accepted that wave I is generated by the auditory nerve action potential. Wave II arises from the cochlear nucleus with wave III at the level of the superior olivary complex. Wave IV represents the lateral lemniscus and wave V, which is generated in the area of the inferior colliculus, is usually the strongest and most identifiable wave. Each of the waves identified above occur at a predictable latency relative to the presentation of the auditory stimulus. The latency values of wave V, relative to the stimulus onset and the latency of wave I, are the most commonly used values for reporting BERA results.

Address reprint requests to Dr. Cummings, 427 No. Hillside, Wichita, KS 67214.

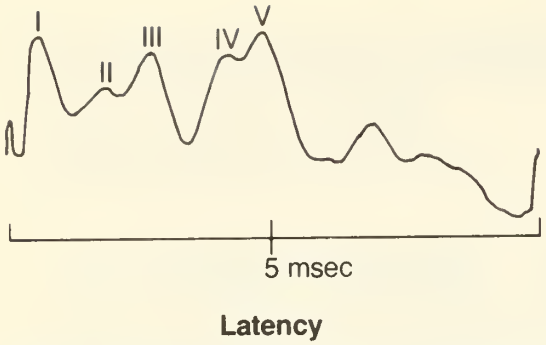


Figure 1. BERA wave configurations for an adult male with normal hearing.

Latency has been found to be affected by stimulus intensity, age, sex, and various pathologies associated with the peripheral hearing system.

It is necessary that the patient remain as stationary as possible throughout the testing procedure. For infants, small children, and other individuals who cannot or will not remain inactive for the test, a mild sedative is administered at least one-half hour before the test begins.

Clinical Applications

In our clinical setting, BERA is used for two principal functions: (1) for hearing threshold estimations; and (2) to diagnose certain central nervous system auditory abnormalities. The greatest portion

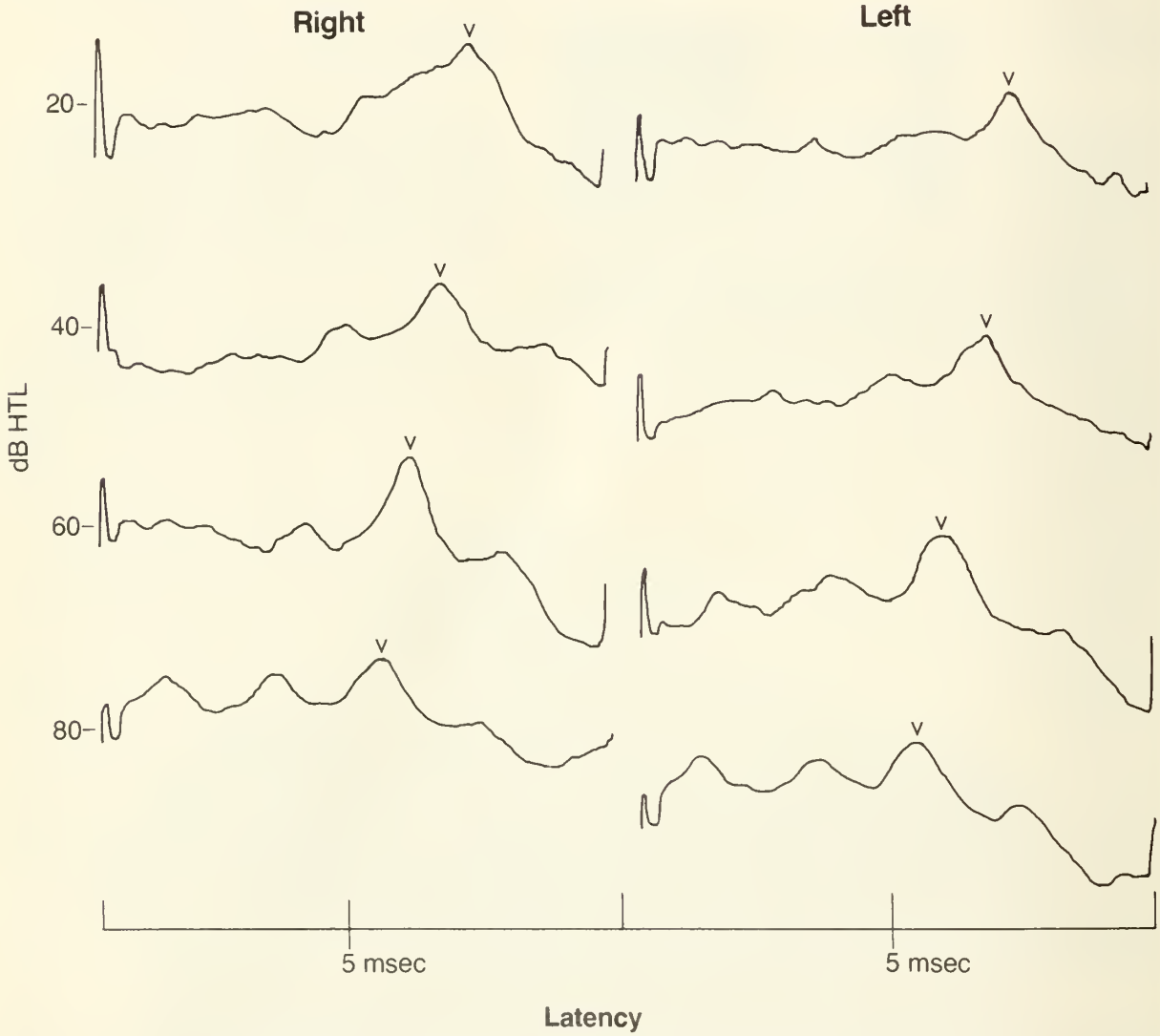


Figure 2. BERA findings in a 12-year-old female suspected of malingering.

of BERA testing is performed for the estimation of peripheral hearing thresholds. This is particularly valuable in the testing of newborns, mentally handicapped children, and children who have not developed language. A newborn in various high-risk categories should have BERA testing between three and nine weeks of age. For newborns, BERA is relatively easy to perform with consistent and reliable results. For patients with Down's Syndrome and other types of retardation, BERA testing is indicated due to the higher incidence of hearing loss and difficulty in testing for this population. BERA is also indicated for the child who has not developed language and is otherwise difficult to evaluate.

BERA has been valuable in our office in detecting malingerers. Often this is a patient who is feigning a hearing loss for compensation purposes following an industrial or automobile accident. The test is also used for otherwise normal youngsters who, for some reason, will not give objective results during routine testing.

Another, less common use for BERA is in evaluating cochlear reserve in a patient using bone-conducted stimuli. A typical example of this would be a youngster with atresia of the external ear canal. Bone conduction BERA is difficult to do and not as accurate as routine air conduction BERA. Where air conduction testing is possible, conductive hearing loss can be identified by comparing latency values between ears and with normative data. A conductive loss will present longer wave V latency when compared to normal ears or ears with sensorineural hearing loss.

Certain central nervous system lesions can cause an abnormality in the BERA. This abnormality may be in the latency interval or in the amplitude of the fifth wave. This is particularly useful in the diagnosis of a small acoustic neuroma. It is felt that BERA is the most sensitive technique for the early detection of an acoustic neuroma.[4] Multiple sclerosis will either present an abnormal fifth wave latency interval or a complete absence of the fifth wave. BERA is useful in helping to diagnose multiple sclerosis when only a solitary lesion is known.[5]

Case Studies

Brainstem evoked response audiometry is now a standard diagnostic tool in otology. It is useful for many purposes, but it is most valuable in assessment of auditory acuity in children and in the diagnosis of acoustic neuroma in adults. The following cases represent some of the ways BERA can be useful in the Otology Clinic.

Case One: Malingering. A 12-year-old female complained of dizziness and decreased hearing. Physical examination of ears, nose, and throat produced negative results. Pure tone hearing testing was attempted, and results indicated inconsistent responses for both ears. Speech reception threshold testing also yielded inconsistent responses. BERA was recommended due to the unreliable results of behavioral audiometric testing. BERA results *(Figure 2)* indicated normal peripheral hearing responses.

Case Two: Assessment of Hearing. A 3-year-old female was referred for BERA with a history of hemophilus meningitis and bilateral hearing loss. Subsequent BERA testing *(Figure 3)* confirmed a profound loss of peripheral hearing sensitivity.

Case Three: Delayed Language Development. A 2-year-old female was brought to us by her parents who were concerned about her slow language de-

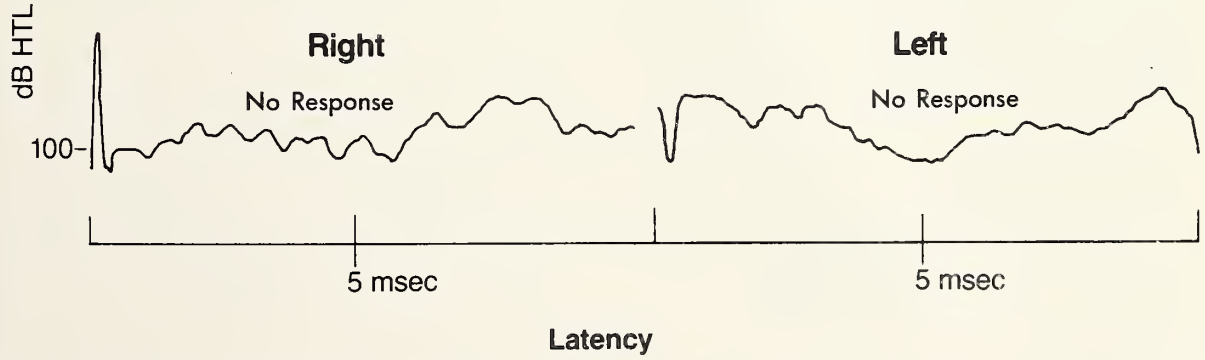


Figure 3. BERA findings showing the absence of identifiable wave configurations obtained in a 3-year-old female.

velopment. The child was generally unresponsive to stimuli presented in a sound field testing situation. Impedance audiometry showed normal middle ear function bilaterally with reflex thresholds present for both ears. BERA results *(Figure 4)* revealed clear responses within the normal hearing range. As with all patients with similar BERA results, the findings were explained to the parents in terms of *peripheral* hearing sensitivity making sure that the parents understood that the test did not indicate how the information was assimilated at higher levels.

Case Four: Retrocochlear Lesion. A 38-year-old male gave a history of constant tinnitus and hearing loss for the left ear. Audiometric testing yielded pure tone averages of 15 dB right and 47 dB for the left ear. Masked speech discrimination for the left ear was 32 per cent. Stapes reflex decay was within normal limits. BERA was performed with logon stimuli presented at 90 dB bilaterally. Results *(Figure 5)* showed the total absence of identifiable wave configurations for the left ear with normal results for the right ear. Internal auditory canal planigrams were performed with findings compatible with left-side acoustic neuroma. The presence of an acoustic neuroma was surgically confirmed with the removal of a 2.5-cm tumor.

Summary

The preceding cases are representative of the variety of patients for whom BERA has been performed. BERA has proven to be an asset to both the otologist and the audiologist. It has provided an efficient means for making a more confident diagnosis in areas where this has previously been difficult or impossible.

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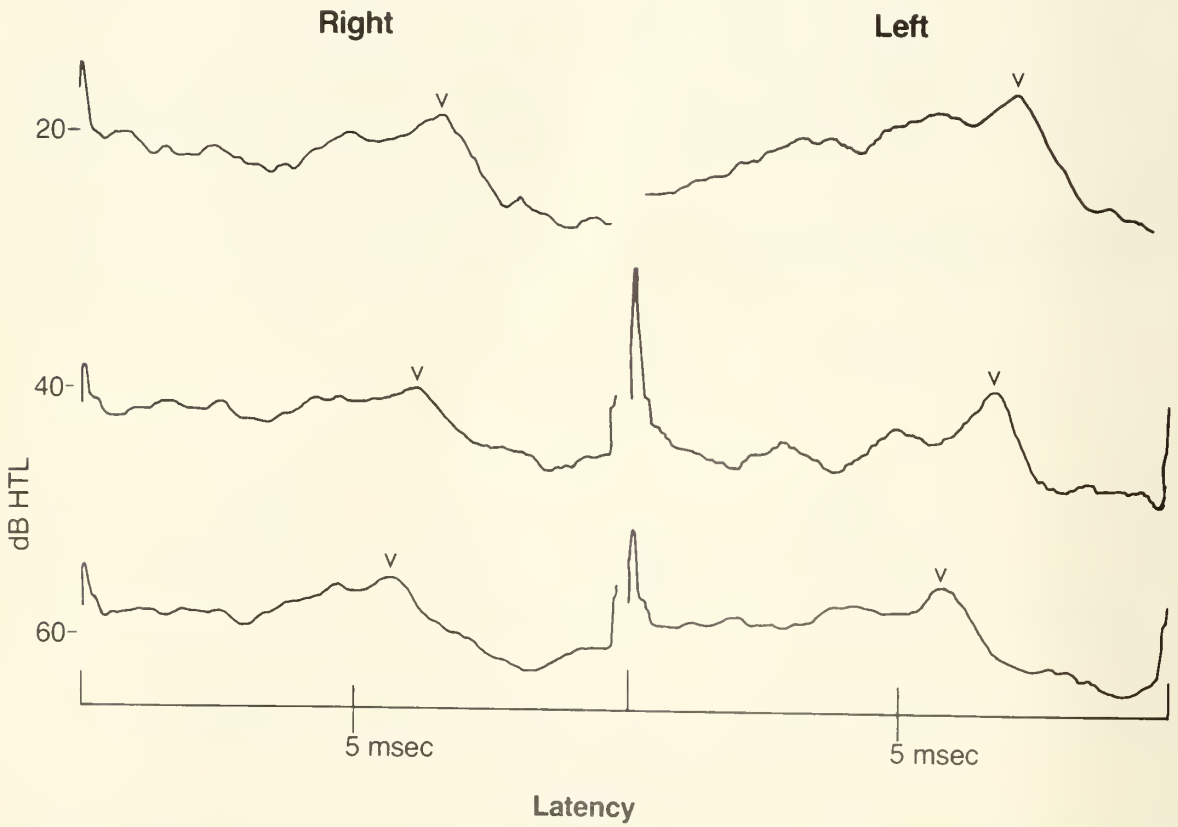


Figure 4. BERA findings in a 2-year-old female with delayed speech and language development.

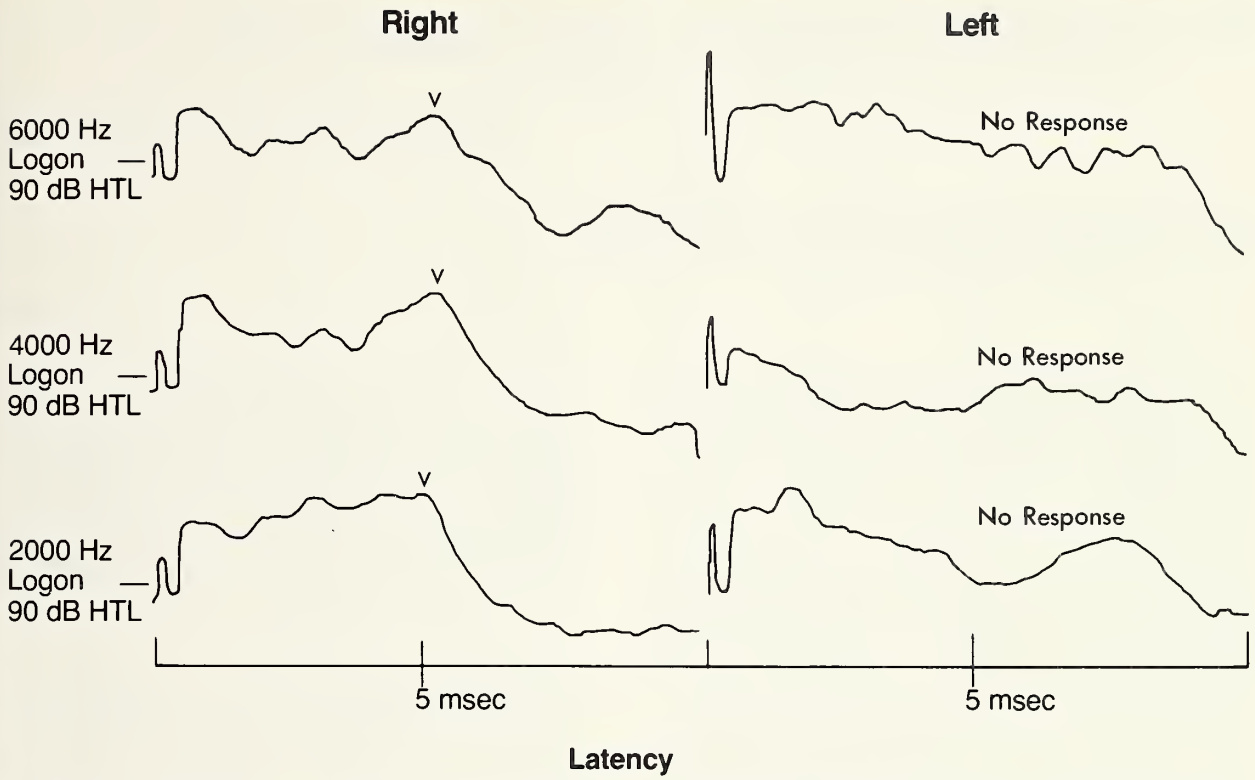


Figure 5. BERA findings in a 38-year-old male prior to surgical removal of a left-side acoustic neuroma.

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Kansas Press Looks at Medicine

(Continued from page 3)

expanding society to inform the public and at the same time protect its interests. Other than through hearsay, it was nearly impossible for one to know what a particular doctor offered or to have any basis on which to base a comparison of physicians.

The proposed legislation seems successfully to handle the delicate balancing act of preserving the doctors' freedom of speech and of informing prospective patients, while protecting the public and maintaining the dignity of the medical profession. — *The Wichita Eagle,* Oct. 22, 1979.

Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

Editor
The Journal of the Kansas Medical Society
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Procainamide-Induced Agranulocytosis

Report of Two Cases and Review

KAVI G. NAGESH, M.D.;* K. P. POULOSE, M.D.;†
TONY H. TSENG, M.D.;‡ *and* ALI SCHADCHEHR, M.D.,§ *Leavenworth*

Two cases of procainamide-induced agranulocytosis are reported in which the patients attained hemotological recovery on withdrawal of the drug and treatment of associated infections. Pathophysiology of procainamide-induced agranulocytosis is discussed and cases analyzed.

PROCAINAMIDE HYDROCHLORIDE is well established as an effective drug in the prophylaxis and treatment of ventricular arrhythmias, especially those following myocardial infarction.[1] It is usually well tolerated. Its known side effects include nausea, vomiting, diarrhea, joint and muscle pain, fever and chills, pruritus, urticaria, and psychosis with hallucinations.[2] Fifty to one hundred per cent of patients on long-term procainamide therapy develop antinuclear antibodies.[3, 4] A smaller percentage of these patients develop a lupus erythematosus-like clinical picture.[3]

Agranulocytosis is a rare but often fatal complication. In world literature, 15 cases of agranulocytosis have been reported in patients receiving procainamide.[5-18] Two more such cases are reported here, bringing the total number to 17 *(Table I)*.

Case One

A 67-year-old white male was readmitted to the hospital on March 20, 1978, with history of increasing shortness of breath, chest pain, swelling of feet and legs, and distention of the abdomen during the week prior to admission. He had been previously hospitalized for similar symptoms from January 6 through February 27, 1978. On February 2, he was noted to have frequent premature ventricular contractions (PVCs). This rhythm disturbance was treated initially by intravenous infusion of lidocaine. Oral procainamide administration was started on February 6 at a dosage of 250-500 mg/4 hrs, and on February 16 the dosage was changed to 500 mg/6 hrs. He received this dosage until he was discharged from the hospital on February 27. He was receiving the same dosage of procainamide when he was readmitted to the hospital.

He was known to have had hypertension for 11 years and had received various antihypertensive drugs with fair-to-good control of blood pressure. At the time of readmission, he was receiving the following oral medications: digoxin, 0.25 mg/day; furosemide, 40 mg/day; potassium chloride 10%, 20 mEq/day; prazosin, 1 mg 2 times/day; alpha methyldopa, 250 mg 4 times/day; procainamide, 500 mg 4 times/day; and isosorbide dinitrate, 10 mg 4 times/day.

Physical examination revealed a well-developed, obese, Caucasian male in no acute distress. Temperature was 37C; pulse, 74 beats/min; and respiration, 30/min. Blood pressure was 160/90 mmHg; head, ears, eyes, nose, and throat were unremarkable except for arcus senilis; jugular veins were engorged. Point of maximal impulse (PMI) was palpable in the left fifth intercostal space in the anterior axillary line. Cardiac rhythm was regular with occasional premature beats. Auscultation of the heart revealed normal S_1, physiological S_2, and faint S_3 gallop over the apex. A Grade II/VI systolic murmur was heard over the left sternal border and apex. Auscultation of the lungs revealed fine rales over both bases. The abdomen was distended due to ascites; the liver was palpable for 9 cm below the costal margin with a vertical span of 15 cm by percussion. The spleen was palpable for 4 cm below the costal margin. There was 3+ pitting ankle and leg edema.

Laboratory Data: Complete blood count (CBC) on March 21 revealed: hemoglobin, 10.2 gm/dl; hematocrit, 31.8%; and white cell count, 1,200/μl with a differential count of 4% segmented neutrophils, 4% basophils, 84% lymphocytes, and 8% monocytes. Peripheral blood smear revealed slight anisocytosis and an adequate number of platelets.

From the Veterans Administration Medical Center, Leavenworth, Kansas.
* Staff physician
† Chief, Medical Service
‡ Chief, Cardiology Section
§ Chief, Gastroenterology Section

TABLE I*
SUMMARY OF 17 CASES OF AGRANULOCYTOSIS FOLLOWING PROCAINAMIDE THERAPY

Author	*Year*	*Age*	*Sex*	*Reason For Use*	*Days of Exposure*	*Daily Dose (g)*	*Total Dose (g)*	*Outcome*
Miller, Et al[5]	1951	70	M	PVC	78	1.0	78	Death
Inouye, et al[6]	1951	87	F	PVC	48	0.75	36.5	Recovery
Antzis, et al[7]	1952	71	F	V Tach	77	0.75-4.5	316.3 Approx.	Death
Fabregoule, et al[8]	1954	68	M	PAT	26	1.5	39	Recovery
Talmers, et al[9]	1965	71	M	Acute MI	78	1.0	78	Recovery
Oster[10]	1966	56	M	Acute MI	30	?	?	Recovery
Wang and Schuller[11]	1969	75	M	PAT	62	0.75-2	113.5	Death
Ream and Barrera[12]	1970[a]	52	M	PVC	90	2.0	180 Approx.	Recovery
Konttinen, et al[13]	1971	75	F	V Tach	17	?	39	Recovery
Konttinen, et al[13]	Same Patient				30	?	69	Recovery[b]
Hickson, et al[14]	1972	45	F	Acute MI	42	1.0	42	Death
Hickson, et al[14]	1972	68	M	Acute MI	33	2.0	66	Recovery
Gunay and Honig[15]	1974	12	F	PVC	13	1-2	?	Recovery
Dross[16]	1974	70	F	V Tach	32	1-2	37	Recovery
Chelius and Nunag[17]	1975	69	M	PVC	90 (Approx)	0.75-1	40.75	Recovery
Prince, et al[18]	1974†	69	F	Atrial Fib	26	1.5-4	57.5	Recovery
Nagesh, et al	1979	67	M	PVC	49	1.5-3	103.5	Recovery‡
Nagesh, et al	1979	80	M	PVC	57	1-2	110	Recovery

* Modified from PRINCE, R. A. *et al.*, American Journal of Hospital Pharmacy 34: P 1363, 1977.

[a] This was the second course of procainamide therapy. The first course which lasted 10 days at a dosage of 2 g/day was accompanied by marked urticaria and itching. The patient was desensitized to the drug with methylprednisolone.

[b] Ten days after procainamide withdrawal, the patient appeared to recover with significant granulocytosis. However, two days later she suddenly died of acute myocardial infarction which was verified at autopsy.

† Published in 1977.

‡ Sudden death on 7/4/78. Autopsy revealed normal bone marrow.

Urinalysis revealed 1+ protein and 2-4 white blood cells/high-power field. Serum chemistry revealed the following values: sodium, 143 mEq/l; potassium, 3.8 mEq/l; chlorides, 104 mEq/l; carbon dioxide, 32.7 mEq/l; BUN, 22.9 mg/dl; creatinine, 1.1 mg/dl; SGOT, 6 IUs/l; SGPT, 6 IUs/l; total serum bilirubin, 0.9 mg/dl; serum protein, 6 gm/dl; and serum albumin, 3.1 gm/dl. Lupus erythematosus (LE) cell preparation was negative. Antinuclear antibody (ANA) titer was negative at 1:20 dilution; rheumatoid activity was negative. Electrocardiogram revealed normal sinus rhythm, occasional PVCs, low voltage in limb leads, left axis deviation, left ventricular hypertrophy, widespread ST segment and T-wave changes consistent with myocardial ischemia, and QS pattern in leads V_1 through V_3 suggestive of old anteroseptal myocardial infarction. Chest x-ray revealed marked cardiomegaly with clear lung fields.

Hospital Course: The patient was initially treated in the coronary care unit with bed rest and a 500 mg sodium diet, and was continued on furosemide, digoxin, alpha methyldopa, procainamide, isosorbide dinitrate, and potassium chloride. Oxygen was administered via nasal cannula at 3 liters/min. He continued to experience chest pain, and the isosorbide dinitrate dosage was increased to 20 mg 4 times/day orally. Nitroglycerin ointment 2% was applied externally at a dosage of 1 in/4 hrs, and the prazosin dosage was gradually increased to 5 mg 2 times/day orally. He was given additional doses of furosemide intravenously. His chest pain was relieved; he had good diuresis with diminution of ankle and leg edema; and his blood pressure was under good control. In view of agranulocytosis, procainamide was discontinued on March 27. However, on the eighth hospital day (*i.e.* March 28), he developed low grade fever. Cultures of blood, throat, sputum and ascitic fluid were sterile. A urine culture grew Enterococcus, group D, more than 100,000 colonies/ml sensitive to gentamicin. The white cell count when he became febrile was 1,100/μl with differential count of 94% lymphocytes, 4% segmented neutrophils, and 2% monocytes. Bone marrow aspiration and biopsy revealed normal erythropoietic tissue and a normal number of megakaryocytes. There was a complete lack of polymorphonuclear cells; however, some promyelocytes, myelocytes, and metamyelo-

TABLE II
COURSE OF BLOOD COUNTS IN CASE 1WITH AGRANULOCYTOSIS

Date	Hgb GM/DL	WBC PER μL	Neutrophils BAND FORMS %	Neutrophils SEGMENTED FORMS %	Eosinophils %	Basophils %	Lymphocytes %	Monocytes %	Platelets PER μL
1/6/78*	13.8	6,900	1	74	3	1	18	3	Adequate
3/21/78	10.2	1,200	0	4	0	4	84	8	Adequate
3/25/78	10.2	900	0	0	0	0	100	0	
3/27/78	10.2	900	0	0	0	0	100	0	231,000
3/28/78	10.7	1,100	0	4	0	0	94	2	260,000
3/31/78	11.0	1,300	1	0	0	1	92	6	Adequate
4/1/78	11.0	2,100	2	3	0	0	87	8	
4/2/78	11.7	2,400	23	5	0	2	65	5	
4/3/78	11.9	6,500	40	16	0	0	36	8	
4/4/78	12.6	9,900	33	19	1	2	36	9	427,000
4/7/78	12.9	18,500	44	30	0	2	14	10	
4/12/78	13.1	16,400	12	68	1	0	10	9	Adequate
4/17/78†	13.4	14,400	4	78	0	0	16	2	Adequate
5/30/78‡	14.0	8,000	6	80	0	0	11	3	Adequate

* During previous admission.
† On the day of discharge.
‡ On subsequent admission.

cytes were seen. He was put in reverse isolation and was treated with a course of gentamicin, 80 mg intramuscularly/8 hrs for ten days. He became afebrile within the next four days (*i.e.* by the 12th hospital day), and remained so until he was discharged from the hospital. Following discontinuation of procainamide, the white cell count — including granulocytes — increased gradually; hemoglobin level also improved. The course of his blood counts is shown in *Table II*.

Since he had hepatomegaly, a liver biopsy was done on April 5; this revealed normal architecture of the liver parenchyma and minimal infiltration of the portal zone with chronic inflammatory cells.

At the time of discharge from the hospital on April 17, the patient was receiving the following medications: spironolactone, 25 mg 4 times/day orally; digoxin, 0.125 mg/day orally; disopyramide phosphate, 100 mg 4 times/day orally; furosemide, 20 mg 2 times/day orally; nitroglycerine ointment 2%, 2 in/6 hrs externally; alpha methyldopa, 250 mg 3 times/day orally; and prazosin, 5 mg 2 times/day orally.

On subsequent hospital admission on May 30, 1978, his blood count was normal *(Table II)*. He was discharged again on June 20.

However, the patient suffered a cardiac arrest on July 4 and was dead on arrival at the hospital. Autopsy revealed a markedly enlarged heart weighing 750 gm with almost complete occlusion of the anterior descending branch of the left coronary artery and also the right coronary artery. The liver weighed 2,500 gm. Bone marrow histology revealed normal myeloid and erythroid series.

Comment: This patient had a normal blood count on January 6, 1978. Procainamide was started on February 6 for control of PVCs; daily dosage varied from 1,500-3,000 mg. He received a total dosage of 103.5 gm over a period of 49 days. He developed agranulocytosis while he was receiving procainamide. Bone marrow studies revealed absence of mature neutrophils and decrease of promyelocytes, myelocytes, and metamyelocytes. The temporal relationship of the onset of agranulocytosis while he was receiving procainamide and the gradual recovery following discontinuation of this agent suggest that procainamide was the drug involved in inducing agranulocytosis. It is of interest to note that he did not have LE cells or significant elevation of ANA.

Case Two

An 80-year-old white male was readmitted to the hospital on December 28, 1978, with a history of sore throat, fever, malaise, body ache, cough, and scanty sputum during the week prior to admission. This was his third hospital admission in 1978, and he had been hospitalized on several occasions during

the preceding six years. He had diagnoses of arteriosclerotic heart disease with angina and episodes of congestive heart failure and calcific aortic stenosis, mild generalized degenerative arthritis, and gout with attacks of acute gouty arthritis. The acute episodes had been treated with short courses of indomethacin in the past, and he had received allopurinol, 300 mg/day for two years, 1972-1974. Allopurinol was restarted in November 1977. He was receiving digoxin and furosemide orally and nitroglycerin sublingually with satisfactory control of congestive heart failure and angina pectoris. He had not received indomethacin during the six months prior to his latest admission in 1978. His immediate previous hospitalization was November 2-18, 1978, for treatment of unstable angina. He was found to have frequent PVCs which were treated by intravenous administration of 100 mg of Lidocaine bolus, followed by intravenous drip of lidocaine at 3 mg/min for the next 24 hours. Oral administration of procainamide was started on November 2 at a dosage of 250 mg/4 hrs. The dosage was increased to 500 mg/6 hrs on November 6. He was also administered Isosorbide dinitrate 10 mg/4 hrs orally, and had external application of 2% nitroglycerin ointment, 1 in/6 hrs. Furosemide and digoxin were continued. The unstable angina improved and he was discharged from the hospital on November 18. He was receiving the following oral medications at that time: digoxin, 0.125 mg/day; procainamide hydrochloride, 500 mg/6 hrs; spironolactone, 25 mg 4 times/day; isosorbide dinitrate, 20 mg/6 hrs; allopurinol, 300 mg/day; and dioctyl sodium sulfosuccinate, 100 mg 2 times/day. He was also receiving external application of nitroglycerin ointment 2%, 1 in/6 hrs. All of these medications were continued until he was readmitted on December 28, 1978.

Physical examination revealed a well-built, moderately-nourished, Caucasian male in some distress due to sore throat. Temperature was 38C; pulse, 72 beats/min; respiration, 26/min; and blood pressure, 100/60 mmHg. He had immature cataracts bilaterally; fundi could not be adequately visualized. He was markedly deaf bilaterally. Tonsils were covered with grayish exudate. Posterior wall of the pharynx was hyperemic; no ulcers were seen on the tongue or buccal mucosa; there was slight enlargement and tenderness of the tonsillar lymph nodes bilaterally. Neck veins were not engorged. PMI was in the fifth left intercostal space 1 cm lateral to the midclavicular line. Auscultation of the heart revealed slight diminution of the intensity of both S_1 and S_2; there was a Grade II-III/VI ejection systolic murmur over the aortic area with conduction to the neck vessels, left sternal border, and cardiac apex. Auscultation of the lungs revealed medium rales over the right base. Examination of the abdomen and extremities revealed no abnormalities. Neurological examination was within normal limits except for marked bilateral deafness.

Laboratory Data: Complete blood count revealed hemoglobin, 13.4 gm/dl; hematocrit, 38.9%; white cell count, 2,800/μl with a differential count of 1% segmented neutrophils, 61% lymphocytes, 3% basophils, and 35% monocytes. Peripheral blood smear revealed adequate platelets and normal red cells. Throat culture grew Candida albicans. Sputum gram stain revealed a few pus cells, moderate gram positive cocci. Culture grew 10% Staphylococcus aureus, 10% Candida albicans, 20% Klebsiella pneumoniae, and 60% normal flora. LE cell preparation was negative. ANA titer was positive at 1:80 dilution. Venereal disease research laboratory test was non-reactive; anti-streptolysin titre and monospot test were negative. Serum complement C_3 was 130 mg/dl (normal range 80-160 mg/dl) and serum complement C_4 was 22 mg/dl (normal range 20-60 mg/dl). Serum chemistry values were normal regarding BUN, creatinine, uric acid, potassium, bicarbonate, SGOT, SGPT, lactate dehydrogenase, alkaline phosphatase, calcium, and total protein. Serum sodium was 132 mEq/l; serum chloride, 95 mEq/l; serum phosphate, 2.4 mg/dl; serum albumin, 3.2 gm/dl; and fasting blood glucose, 138 mg/dl. Urinalysis was unremarkable, and urine culture was negative. Chest x-ray revealed infiltrates in the right lower lung field. Electrocardiogram revealed regular sinus rhythm, right bundle branch block, left atrial enlargement, and an old inferior wall myocardial infarction.

Hospital Course: On receiving the initial blood count, which revealed leukopenia with agranulocytosis, procainamide and allopurinol were discontinued; the patient's other medications were continued as before. He was administered erythromycin, 500 mg/6 hrs orally, and nystatin suspension, 500,000 units to be gargled and swallowed/6 hrs. Bone marrow aspiration and biopsy revealed absence of band forms and segmented neutrophils and reduction in the myeloid series. However, immature cells, including metamyelocytes, were present. The erythroid series and megakaryocytes were normal. He had mild elevation of temperature during the first four days of hospitalization after which he remained afebrile. Exudate on the tonsils due to Candida infection cleared completely by the eighth hospital day, and the lungs became clear to percussion and auscultation. Blood counts were monitored

TABLE III
COURSE OF BLOOD COUNTS IN CASE 2 WITH AGRANULOCYTOSIS

Date	Hgb GM/DL	WBC PER μL	Neutrophils BAND FORMS %	Neutrophils SEGMENTED FORMS %	Eosinophils %	Basophils %	Lymphocytes %	Monocytes %	Platelets PER μL
11/3/78*	13.6	6,700	9	64	3	0	19	5	Adequate
12/28/78	13.4	2,800	0	1	0	3	61	35	Adequate
12/30/78	12.8	3,000	0	0	0	0	58	42	
12/31/78	12.9	3,300	0	0	0	0	76	24	
1/1/79	13.6	2,900	0	0	0	2	78	20	Adequate
1/3/79	13.6	2,800	0	0	0	0	86	14	
1/5/79	13.5	3,400	0	1	0	2	70	27	
1/8/79	14.1	3,600	0	1	0	2	70	27	
1/11/79	14.7	3,500	2	7	0	0	73	18	Adequate
1/14/79	14.9	4,900	7	29	0	0	55	9	
1/16/79†	14.9	5,900	1	70	0	0	27	2	219,000
3/6/79‡	13.9	6,300	2	59	3	2	29	7	Adequate

* Previous admission.
† On day of discharge.
‡ On subsequent admission.

closely. There was practically a total absence of neutrophils until the eleventh hospital day; subsequently, white cell count rapidly returned to normal levels. The course of his blood counts is shown in *Table III*. The patient was discharged from the hospital on January 16, 1979, and was receiving the following medications: digoxin, 0.125 mg/day orally; isosorbide dinitrate, 20 mg 4 times/day orally; spironolactone, 25 mg 4 times/day orally; dioctyl sodium sulfosuccinate, 100 mg 2 times/day orally; and nitroglycerin ointment 2%, 1 in/6 hrs externally. Repeat blood count run on March 6, 1979, was normal.

Comment: This 80-year-old patient with arteriosclerotic heart disease was started on procainamide on November 2, 1978, and had received a total dosage of 110 gm of the drug during a period of 57 days. He developed agranulocytosis while he was receiving procainamide. He was on other medications including allopurinol which is known to produce agranulocytosis; however, he had received this drug for two years, 1972-1974, and also from November 1977 through December 1978. The procainamide had been started for the first time on November 2, 1978. He presented with classical features of agranulocytosis with fever, malaise, and sore throat approximately seven weeks following initiation of procainamide therapy. Prompt withdrawal of the drug and treatment of associated infections resulted in complete hematologic recovery. He had a positive ANA titer at 1:80 dilution, but this is probably not significant in a man 80 years old.

Discussion

Drug-induced agranulocytosis is a severe selective neutropenia resulting from an idiosyncratic reaction to a drug. This form of neutropenia should be distinguished from a similar hematological picture regularly observed following large doses of cytotoxic drugs or bone marrow irradiation.[19]

Drug-induced agranulocytosis is thought to result from one of two basic mechanisms. The first is immunological. Following a period of sensitization, antibodies to drug or drug cell membrane conjugates are formed and ultimately result in neutrophil destruction.

The classic example of a drug causing immunological agranulocytosis is aminopyrine. There is evidence that this mechanism is also operative in at least some cases of disorders due to other drugs, such as sulfonamides, quinidine, and anti-thyroid drugs.[19]

The second mechanism, that of direct bone marrow suppression, is best exemplified by agranulocytosis induced by phenothiazines.

The mechanism of development of agranulocytosis with procainamide remains undetermined, and no data are available to permit the understanding of the reaction in any case.[20] Unless agranulocytosis is a part of lupoid reaction, there does not

appear to be a clear-cut time/dose relationship.[14] No leukoagglutinins have been demonstrated.[10] Sensitization does not appear to be the mechanism since in one case the patient tolerated a larger dose over a longer period of time before developing agranulocytosis for the second time.[13] It has been suggested that ortho- and para-quinone, both breakdown parts of benzamine structures such as para-amino-benzoic acid, may play a role in agranulocytosis and the myelotoxic effect of procainamide may be related to inhibition of nucleic acid synthesis in developing bone marrow.[21]

Many patients receiving procainamide for a long enough period of time develop positive antinuclear reaction, even in the absence of significant symptoms. Only a few eventually sustain illness, which generally takes one of two forms. In the first of these, lupus-like abnormality develops, characterized by arthralgia, chills, fever, skin rash, leukopenia, hemolytic anemia, lymphadenopathy, and hepatosplenomegaly. All of these manifestations clear promptly when the drug is discontinued. The other form is agranulocytosis, which is usually precipitous and frequently associated with infection. As in all cases of agranulocytosis, infection, fever, chills, and pharyngitis are prominent clinical manifestations. Hematologically, there is a complete absence of neutrophilic polymorphonuclear cells from the peripheral blood, but persistence of granulocyte precursors in the bone marrow.[22]

A review of 17 cases — including the two cases in this report — provides some insight into this rare complication of procainamide therapy. There were 10 males and 7 females of ages ranging from 12 to 87 years, with a mean of 65 years. Even though procainamide was discontinued in all of the patients, four (24%) succumbed from the complications of agranulocytosis. Among those who succumbed, there were two females (ages 42 and 77 years) and two males (ages 62 and 78 years). There was no mean age difference between those who succumbed and those who survived. Duration of exposure to the drug ranged from 13-90 days, and the total dosage ranged from 36.5-316.3 gm. There were a total of 18 occurrences in 17 cases (in one case, the patient had agranulocytosis for a second time[13]). Mean duration of exposure among those who succumbed was 64.7 days compared to 43.8 days in those who survived. Although it appeared that a higher cumulative dosage adversely affected the outcome (two of the four patients who died received a total dosage of greater than 100 gm), this does not seem to be absolute, since one patient receiving 180 gm survived.

It is of interest to note that no death due to procainamide-induced agranulocytosis has been reported since 1973. Increased awareness of the complication, which is essentially reversible on withdrawal of the drug and appropriate treatment of infections, may have contributed to the successful outcome in recent years.

Procainamide is metabolized by the hepatic n-acetyltransferase system. Patients who are genetically-determined slow acetylators develop a positive ANA test sooner and SLE syndrome more frequently than do rapid acetylators.[23] Study of a recent case report indicates the efficacy of n-acetylprocainamide (NAPA) in inducing remission of procainamide-induced lupus erythematosus.[24] Further studies are needed to determine whether NAPA is free from the rare adverse effect of agranulocytosis.

Conclusion

Agranulocytosis is an uncommon but potentially fatal complication of procainamide therapy. It is suggested that all patients receiving procainamide should have: (1) a warning to report immediately on development of new symptoms, including soreness of mouth, throat, gums, fever, or symptoms of upper respiratory infection; (2) weekly monitoring of hemoglobin and white cell counts — both total and differential — during the first three months, since all reported cases have occurred within this time; and (3) routine testing for antinuclear antibodies.

Summary

Two cases of procainamide-induced agranulocytosis are reported. In both cases, patients were exposed to procainamide prescribed for control of premature ventricular contractions associated with arteriosclerotic heart disease. In the first case, the patient, aged 69 years, was exposed to procainamide for 49 days with daily dosage ranging from 1,500-3,000 mg, and had received a total dosage of 103.5 gm. In the second case, the patient, aged 80 years, was exposed to procainamide for 57 days with daily dosage ranging from 1,000-2,000 mg, and had received a total dosage of 110 gm. Complete hematological recovery was accomplished in both cases on withdrawal of the drug and treatment of associated infections.

Pathophysiology of drug-induced agranulocytosis has been briefly discussed and the literature of reported cases of procainamide-induced agranulocytosis analytically reviewed.

Patients receiving procainamide should be closely observed and instructed to report immediately in the event of development of any new symptoms, and

should have weekly monitoring of white cell counts and hemoglobin, and routine testing of antinuclear antibodies.

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Book Reviews

(Continued from page 9)

last four centuries of empiric investigative techniques. The Mayo study, alluded to above, establishes a similar symptomatic benefit for placebo and vitamin C, but once again no survival benefit.

What conclusions can one draw from Dr. Newbold's book? First, the evangelistic fervor of the author will accomplish little in defining the role of vitamin C in cancer treatment, although its antiestablishment tone may accomplish the publisher's intended commercial appeal. The author obviously has no intention of testing his opinions in a scientifically creditable fashion, since that might damage his maverick status. Secondly, his book makes a passing attempt at objectivity, and should not be disregarded in its appeal to increase our scientific investigation of the role of nutrition in the treatment of patients with advanced malignancy. Lastly, although Dr. Newbold would have his readers believe otherwise, the practicing physician can still separate scientifically firm facts from anecdotal enthusiasms. — *R.L.S.*



Current COMMENT

Chronic Lymphocytic Leukemia and Related Lymphoproliferative Disorders

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CHRONIC LYMPHOCYTIC or lymphatic leukemia (CLL) is the most common leukemia encountered by the practicing physician. This paper reviews the current status of the disease and discusses briefly the two rare, but closely related, lymphoproliferative disorders that should be carefully separated from CLL because of important differences in the therapeutic approach.

Chronic Lymphocytic Leukemia

CLL accounts for 25 per cent of all leukemias. It is a disease characterized by malignant proliferation of morphologically mature-looking B-lymphocytes (occasionally T-lymphocytes) which progressively accumulate and obliterate the lymphoreticular network. Peripheral blood, bone marrow, lymph nodes and spleen are primarily involved, and extension to extra-lymphatic organs may occur. It is more frequent in the elderly (median age — 60 years) and occurs twice as often in males as in females. Although clustering of cases in close relatives has been reported, it usually is distributed sporadically. Radiation and chemicals do not seem to have causative significance.

The clinical presentation of CLL is variable. In 25 per cent of patients, the diagnosis is made accidentally from routine blood study. Symptoms and signs that often indicate active disease include fatigue, decreased exercise tolerance, fever, night sweats, weight loss, lymphadenopathy, and splenomegaly. As bone marrow insufficiency develops, anemia, bleeding, and infection become evident.

Diagnosis

As a rule, the diagnosis of CLL is an easy task. The peripheral blood shows an absolute and sustained increase in lymphocytes, usually exceeding 15,000. The majority of the lymphocytes are small and mature-looking, but a few are large and may show a variable degree of atypia. Bone marrow aspiration often yields hypercellular particles, and microscopic examination reveals diffuse infiltration by lymphocytes, morphologically identical to those in the peripheral blood. In early CLL, the involvement of the marrow may be patchy, making histological distinction from benign follicles difficult. In such instances, the diagnosis of CLL rests on careful examination of a biopsy specimen which frequently demonstrates the disruption of normal marrow architecture by infiltrating lymphocytes. Finally, although marrow examination is always a useful baseline study, it may not be necessary for the diagnosis of CLL if the peripheral lymphocytosis is high (roughly $>$ 50,000).

Differential Diagnosis

Disorders that are capable of evoking intense lymphocytic response (*e.g.* tuberculosis, brucellosis, and certain solid tumors) should be excluded by appropriate studies. In patients whose lymphocyte count is below 5,000, but who are suspected of having CLL, an extended period of observation may be required to establish the diagnosis. Alternatively, immunological surface-marker analysis of lymphocytes may provide evidence for early CLL. The characteristic feature of B-cells is the presence of

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surface-bound immunoglobulin, easily detectable by immunofluroscent technique. Since CLL is a neoplasm arising from single clones of B-cells, the surface-marker study shows a uniform pattern of immunoglobulin, restricted to one heavy chain class and a single light chain type.

Lymphosarcoma cell leukemia (LSCL) is a term used to describe the leukemic phase of all lymphocytic lymphomas excluding the well-differentiated sub-type. The cell of LSCL is large. The prominent nucleus is usually folded or clefted, shows variable clumping of chromatin, and contains conspicuous nucleolus. As a rule, confirmation of the disease is obtained from lymph node biopsy. Finally, the resemblance of CLL to prolymphocytic leukemia and hairy-cell leukemia may be close. Because of the substantial difference in treatment, these disorders will be discussed in some detail below.

Complications of CLL

Anemia occurs commonly and is primarily due to inadequate bone marrow production. Although the pathogenesis of the depressed erythropoiesis is not fully known, "over-crowding" of the marrow may be an important contributing factor. Coomb's test is positive in 20-25 per cent of CLL patients, but less than 5 per cent develop clinically significant hemolytic anemia. As a rule, thrombocytopenia indicates a more advanced disease, and its severity correlates with the degree of bone marrow failure. Immune thrombocytopenia with accelerated platelet destruction is a rare complication.

Since CLL is a B-cell dyscrasia, abnormality of immunoglobulin production is predictable. Hypogammaglobulinemia occurs in 50 per cent of patients and becomes more severe with progression of the disease. It contributes to increased incidence of infection, the leading cause of death in CLL. Less than 5 per cent of patients develop monoclonal gammopathy, commonly IgM, which sometimes makes the separation from Waldenström's macroglobulinemia difficult. Hyperuricemia, which is present in 30 per cent of patients, is rarely associated with clinical gout. In contrast to chronic granulocytic leukemia, CLL rarely undergoes blastic transformation.

Treatment

The optimal management of the individual patient with CLL is controversial. However, the mere diagnosis of the disease does not justify treatment. An accepted strategy is to withhold systemic therapy until there are signs and symptoms of progressive disease. Though disagreement still exists, the indices of disease activity summarized in *Table I* serve as acceptable guidelines. It should be noted that the white cell count per se is not an accurate indicator of the extent or activity of the disease.

The major treatments for CLL include alkylating agents, corticosteroids, and irradiation. Chlorambucil (CLB) and cyclophosphamide (CY) are the most widely used alkylating agents, and the usual induction dosages are: CLB, 0.1-0.2 mg/kg/day; and CY, 2.0 mg/kg/day. For successful use of chemotherapy, it is essential to do frequent blood counts and make the appropriate adjustment in dosage as required. A useful guideline is to reduce the dosage by 50 per cent as the white cell count (WBC) reaches half of the starting dose. The WBC may take up to three months to reach a level below 15,000. Although CLB and CY are equally effective, the "platelet-sparing" effect of CY may be an advantage in treating thrombocytopenic patients.

Corticosteroids are commonly given in combination with an alkylating agent. Short-term steroid therapy has been beneficial in patients who are unable to take myelosuppressive drugs because of severe bone-marrow failure. In such cases, prednisone, 40-60 mg/day for two to four weeks, may improve the marrow status significantly and eventually allow the addition of an alkylating agent. Furthermore, corticosteroids have an established role in the treatment of symptomatic hemolytic anemia and immune thrombocytopenia.

The use of intermittent high dose therapy with an alkylating agent and a steroid (*e.g.* CLB: 0.4-0.8 mg/kg with short-course prednisone every 28 days) has sound kinetic principal. It is well tolerated and simple to administer. Moreover, it is as effective and may at times be useful in patients who are refractory to continuous low-dose therapy.

The limited experience gained with whole body irradiation (WBI) is encouraging. However, recommendations for its wider use should be determined by the outcome of a prospective randomized

TABLE I
INDICATIONS FOR TREATMENT OF CLL

A. Symptoms attributable to CLL: fever, night sweats, weight loss, impaired exercise tolerance.
B. Presence of bone marrow insufficiency: progressive anemia, thrombocytopenia.
C. Evidence of progressive infiltrative disease: lymphadenopathy, splenomegaly, etc.
D. Immune hemolytic anemia or immune thrombocytopenia or both.

study comparing WBI to chemotherapy. Involved field radiotherapy provides an effective approach for control of localized disease (*e.g.* lymphadenopathy causing cosmetic or local mechanical problems).

When splenomegaly, unresponsive to systemic chemotherapy, becomes symptomatic (cytopenia, discomfort), splenic irradiation or splenectomy may be tried. Unfortunately, splenic irradiation has sometimes resulted in life-threatening thrombocytopenia, and surgical intervention always carries a substantial risk in elderly patients.

Future Trends

Based on clinical and laboratory findings, Rai *et al.* have proposed a four-stage classification of CLL *(Table II)*. While the overall survival time in CLL is 71 months, Stages III and IV have survival times of 19 months — significantly shorter than the earlier stages. It is hoped that the wider application of such or similar classification will allow better stratification of patients and meaningful comparison between different modalities of therapy.

So far, the aim of treatment in CLL has been generally palliative since its impact on long-term survival has been minimal at best. In recent years, however, the preliminary data obtained on selected patients who received aggressive combination chemotherapy has been encouraging. With the advent of a better staging system, it will be possible to identify an increasing number of patients who may benefit from aggressive therapy, with a goal of cure rather than palliation. However, in as much as CLL is a disease of the elderly, the advantage of cure must be balanced against the risks of intensive therapy. Finally, there are early studies that suggest a correlation between the specific class of surface-bound immunoglobulin and the clinical progression of the disease. This may provide an additional criterion in the selection of patients for intensive treatments.

Prolymphocytic Leukemia (PLL)

PLL is a rare variant of CLL with distinct clinical features. It occurs mainly in the elderly and preferentially affects males. The majority of the patients are symptomatic — weight loss, fever, sweats, fatigue, and weakness being common presenting complaints. On physical examination, marked splenomegaly is a very common feature. The liver may be variably enlarged, and adenopathy is uncommon. Frequently, there is a striking elevation in the WBC with a median value of 350,000/μl. The abnormal cell (prolymphocyte) is larger than the small lymphocyte and has moderately abundant cytoplasm. Its nucleus has coarse chromatin and a prominent nucleolus (the morphological distinction from LSCL is at times difficult). The prognosis of PLL is poor with a median survival time of 48 weeks. It is usually refractory to a single agent chemotherapy or corticosteroids. Leukophoresis, splenectomy, or both have been helpful in some patients. The data on combination chemotherapy is still anecdotal.

TABLE II
STAGES OF CLL AND CORRELATION WITH SURVIVAL

Stage	*Finding*	*Median Survival*
0	Blood and bone marrow lymphocytosis only	150 mos
I	Lymphocytosis with enlarged nodes	101 mos
II	Lymphocytosis with enlarged liver and/or spleen	71 mos
III	Lymphocytosis with anemia, ± enlarged liver and/or spleen	19 mos
IV	Lymphocytosis with thrombocytopenia, ± enlarged liver and/or spleen	19 mos

Hairy-Cell Leukemia (HCL)

HCL or leukemic reticuloendotheliosis is a rare lymphoproliferative disorder which usually presents with cytopenia and splenomegaly without lymphadenopathy. It has male predominance, and the mean age at diagnosis is 50 years. The identifying feature is the presence of mononuclear cells with fine cytoplasmic projections (hence the name HCL) in the peripheral blood, marrow, and spleen. Typically the mononuclear cells contain a high percentage of tartaric acid-resistant acid phosphatase. The disease may have an indolent course, and infection — primarily related to leukopenia — is the leading cause of death. Most patients benefit from splenectomy which is the treatment of choice for symptomatic cytopenia. It improves the quality of life, and perhaps prolongs survival. Chemotherapy with single alklyating agent is reserved for patients who have progressive disease despite splenectomy. The cytoxic drug should be administered with close supervision of the blood count because of the preexisting bone marrow insufficiency. The use of combination chemotherapy for aggressive disease refractory to conventional treatment is yet to be evaluated.

Summary

Table III provides a concise delineation of clinical

TABLE III
SELECTED CLINICAL FEATURES AND TREATMENT OF CHRONIC LYMPHOCYTIC LEUKEMIA, PROLYMPHOCYTIC LEUKEMIA, AND HAIRY-CELL LEUKEMIA

Disorder	*Age/Sex*	*Lymphadenopathy*	*Splenomegaly*	*Treatment*
CLL	> 40 M > F	+ to ++	+ to +++	Alkylating agent corticosteroids
PLL	> 60 M > F	±	+++	Leukophoresis splenectomy
HCL	> 30 M > F	±	+++	Splenectomy

(−) not present; (+) Mild; (++) Marked; (+++) Massive.

features and treatment of the three leukemias discussed here.

CLL is a lymphoproliferative disorder characterized by malignant proliferation and progressive accumulation of immunoincompetent B-cells. It is the most frequent form of leukemia in the Western world, affecting primarily elderly males. Peripheral blood, bone marrow, lymph nodes, and spleen are most commonly involved, and as a rule the diagnosis is readily apparent. Some patients with CLL have a benign and indolent course, but the majority develop progressive disease and die of their leukemia. Presently therapeutic intervention is determined by the presence or absence of progressive disease *(Table I)*. The mainstay of therapy consists of alkylating agents and corticosteroids, administered either continuously or intermittently. Local irradiation is effective in the control of localized disease, but the role of WBI is yet to be determined. Since the recently developed staging system correlates well with survival, it promises to provide a better comparison between different treatment trials and to allow the selection of proper treatment for individual patients.

PLL is a rare disease of the elderly male, characterized by high leukocyte count — almost all prolymphocytes — and massive splenomegaly. The prognosis is poor, but some patients have benefited from leukophoresis or splenectomy or both.

The main manifestations of HCL are pancytopenia and splenomegaly, and the diagnosis is usually confirmed by the presence of "hairy cells" which typically contain tartarate-resistant acid phosphatase. The treatment for symptomatic cytopenia is splenectomy, and if this fails, cautious trial with a single alkylating agent is recommended.

Self-Assessment Questions

1. Which of the following statements are applicable to chronic lymphocytic leukemia?
 a. Hypogammaglobulinemia is infrequent.
 b. It commonly undergoes blastic transformation.
 c. Treatment should be started when diagnosis is made.
 d. Thrombocytopenia correlates with degree of bone marrow failure.
2. A 70-year-old male was admitted for prostatectomy. Physical examination revealed several discrete lymph nodes. The hemogram revealed: hgb, 13 gm/100 ml; WBC, 80,000/μl with 80% lymphocytes; platelets, 150,000/μl. The diagnosis of CLL was established from the peripheral smear study. Which of the following statements would be true of the disease?
 a. About 25 per cent of the patients will have positive Coomb's test.
 b. It may follow an indolent course.
 c. Bone marrow study is always necessary to establish the diagnosis.
 d. The level of WBC is an indication to start treatment.
3. Select the statements most applicable to hairy-cell leukemia:
 a. Splenomegaly is an uncommon feature.
 b. Pancytopenia is frequent.
 c. The abnormal cells contain tartarate-resistant acid phosphatase granules.
 d. The treatment of choice is aggressive chemotherapy.
4. Select the statements most applicable to prolymphocytic leukemia:
 a. It has better prognosis than CLL.
 b. It is often refractory to chemotherapy.
 c. Splenomegaly is a common feature.
 d. Marked leukocytosis is infrequent.

(Answers on page 38)

Abstracts

Presented at the 1979 Annual Meeting, Kansas Chapter, American College of Physicians

ALLERGY AND IMMUNOLOGY

Goodpasture's Syndrome Responding to Combination Plasmapheresis and Immunosuppressive Therapy

Robert L. Ricci, M.D.; Robert Weber, M.D. and Arnold Chonko, M.D., UKSM-KC

A 21-year-old white male noted the insidious onset of mild hemoptysis in the fall of 1977. In January 1978, he was hospitalized in Salina for progressive dyspnea on exertion. Bilateral pulmonary infiltrates were detected on chest x-ray, and bronchoscopy revealed iron-laden macrophages. An iron deficiency anemia was present. A diagnosis of Goodpasture's syndrome was made. The patient's work environment had resulted in significant exposure to hydrocarbon solvents. Pulmonary infiltrates transiently improved with rest and his removal from the work environment.

He was referred to UKSM in March 1978, when azotemia developed. Significant laboratory data at that time included: hemoglobin, 8.5 mg/dl; creatinine, 2.6 mg/dl; persistent microscopic hematuria; and proteinuria, 6-15 gm/day. Percutaneous renal biopsy revealed a generalized proliferative glomerulonephritis. Immunofluorescent stains revealed linear deposits of IgG and IgM along the glomerular capillaries. Circulating anti-glomerular basement antibodies were present in high titer.

Simultaneous plasmapheresis and immunosuppressive drug therapy was instituted because of persistent anemia and progressive decline in renal function. The patient's anemia and renal function concurrently improved as circulating anti-GBM-ABs were removed from his plasma. It appears that plasmapheresis, in addition to combination prednisone and cyclophosphamide, is effective for the initial treatment of Goodpasture's syndrome when anti-GBM-ABs are present.

ENDOCRINOLOGY & METABOLISM

Oligohypopituitarism and the Empty Sella Syndrome

Nirandon Wongsurawat, M.D., Veterans Administration Medical Center, Topeka

Endocrine abnormalities in patients with the empty sella syndrome are rare, and are usually mild and without apparent clinical consequences. Of the very few reported cases, hypothyroidism in patients with the empty sella syndrome was either of primary type or was a part of panhypopituitarism. This case is a rare example of thyrotropin-deficient hypothyroidism.

A 79-year-old white male was admitted complaining of numbness in both hands and feet. Results of physical examination were normal except for decreased tactile and pain sensation in the hands and feet. Because of the findings of bilateral carpal tunnel syndrome, thyroid function tests were performed. Serum thyroxine was 2 mEq/dl; resin uptake test was 40.8% (35-45%); and ^{131}I thyroid uptake was 3% at 24 hrs. Serum thyrotropin was 2.9 microU/ml and did not rise after intravenous thyrotropin releasing hormone (TRH). Skull x-ray revealed an enlarged sella turcica. Pituitary tumor was suspected at this point. Serum prolactin was 4.1 ng/ml (0-28) and did not respond to TRH. Visual fields were normal. Computed tomography scan revealed an empty sella containing cerebrospinal fluid as well as a well-defined pituitary. Further endocrine studies — including insulin tolerance test, cortisol levels, corticotropin levels, metyrapone test, testosterone and gonadotropins levels — yielded normal results. The patient did well and the carpal tunnel syndrome resolved after thyroxine replacement therapy. Conclusion: (1) a rare case of thyrotropin deficient hypothyroidism is presented; (2) the empty sella syndrome and secondary hypothyroidism may coexist without causal relationship; and (3) enlarged sella turcica with partial pituitary insufficiency does not always indicate the presence of a tumor; the sella may be half-empty.

Neuroblastoma, Pheochromocytoma, and Renal Cell Carcinoma Occurring in a Single Patient

Richard S. Fairchild, M.D.; Joseph L. Kyner, M.D. and Neil Schimke, M.D., Kansas City

Neuroblastomas and pheochromocytomas are related to one another by their common neural crest origin and ability to synthesize and metabolize catecholamines. Pheochromocytomas and renal cell carcinoma can both occur as manifestations of von Hippel-Lindau disease. However, there are no reports in the literature of a separate neuroblastoma and pheochromocytoma occurring in the same patient; and an individual with von Hippel-Lindau rarely, if ever, has been reported to have both a pheochromocytoma and renal cell carcinoma. The following is the first report of a patient with all three tumors.

A 26-year-old white female had been found at the age of nine months to have a neuroblastoma of the left para-aortic region near the aortic bifurcation which was resected and treated with radiotherapy. She remained free of disease until the age of 16 years when she was found to have a right-sided extra-adrenal pheochromocytoma, which was locally resected. At the age of 21 years, she was noted to be hypertensive with elevated urinary VMA, and a solitary recurrence of the pheochromocytoma was found in the liver and again locally excised. At age 26 years she again became hypertensive and multiple sites of recurrence were noted in the liver. In addition, at least five loci of renal cell carcinomas were found in the left kidney, requiring complete nephrectomy.

Neither this patient nor her immediate family has any ocular evidence to support von Hippel-Lindau disease. The patient's CAT scan was negative. Although her mother is hypertensive, urinary VMAs and IVPs in all family members were negative.

The rare combination of these three tumor types in this single patient appears to be unrelated to a familial syndrome.

Diabetic Honeymoon Stage After Ketoacidosis in an Adult

N. Wongsurawat, M.D.; G. L. Monto, M.D. and V. Wongsurawat, M.D., Topeka

In juvenile diabetes, the presenting symptoms are those of coma or stupor (diabetic ketoacidosis). With appropriate therapy, a period of improvement in carbohydrate tolerance occurs early in the initial treatment of practically all diabetic children. Infrequently the need for exogenous insulin may then disappear for a short time. This so-called honeymoon stage is exceedingly rare in adults.

An obese 72-year-old black male was admitted in a semicomatose state. There was no history of diabetes. The only medication was a diuretic, taken only irregularly, for hypertension. There was a history of increased thirst and polyuria for one week. The patient was markedly dehydrated, pale, and afebrile. Respiration was rapid but shallow. Initial laboratory results: hemoglobin, 11.3; WBC, 18.4/cu mm; neutrophils, 75%; lymphocytes, 25%; glucose, 1155 mg/dl; Na, 120; K, 2.9; Cl, 62; CO_2, 4; BUN, 46; PO_4, 0.5; arterial blood pH, 7.15; pO_2, 146; pCO_2, 9.5; and actual bicarbonate 3 mEq/l. Treatment included IV fluid, K, PO_4, Na_2CO_3, and IM insulin hourly. The patient received 180 units of insulin in 24 hours. Biochemical improvement was gradual over a 36-hour period; however, he remained stuporous for three days. There was no sign of increased intra-

cranial pressure. During the first week, there was gastrointestinal hemorrhage requiring blood transfusion. After six weeks in the hospital, he was discharged on 20 units of Lente insulin daily. On out-patient follow up, his insulin requirement had decreased and the insulin was totally discontinued. The patient remained in remission, asymptomatic euglycemic, for several months. On a recent visit, his plasma glucose had begun to climb and was reported as 170 mg/dl. He had also gained more than 20 lbs since discharge. Conclusion: there can be a period of remission after diabetic ketoacidosis in adults.

^{47}Ca Absorption in Patients Taking Chronic Anticonvulsant Therapy

Timothy O. Wahl, M.D. and Barbara P. Lukert, M.D., UKSM-KC

Chronic phenytoin therapy is known to be associated with osteomalacia. The purpose of this study was to evaluate calcium absorption in 12 patients (seven males, five females) taking anticonvulsants. All 12 were taking phenytoin; eight, phenobarbital; five, primidone; and three, carbamazepine. All patients were fasted for 12 hours and then given 3μCi of ^{47}Ca intravenously. Blood was obtained every 30 minutes for two hours. At this time 15μCi of ^{47}Ca was given orally in ordinary milk vehicle. Blood was obtained every two hours for eight hours. The two radioactive curves were compared and fractional intestinal calcium absorption (FCaA) calculated by the following formula:

$$FCaA = \frac{a}{b} \times \frac{B}{A} \times 100$$

where A is ingested dose, B is injected dose, a and b are points found on the two curves. Following are the results expressed as mean ± SEM.

FCaA subjects	FCaA controls
30.8 ± 3.7%	52.3 ± 3.5%

($p < .005$)

Serum phenytoin levels in the subjects were 12.7 ± 2.5μg/ml; phenobarbital, 15.1 ± 2.5μg/ml; 25-OH-D_3, 30.5 ± 3.6 ng/ml (N 15.0-45.0). Calcium determinations were 9.4 ± 0.1 mg/dl; phosphorous, 3.7 ± 0.1 mg/dl; alkaline phosphatase, 5.6 ± 0.6 BL units (N 1.5-6.3); parathyroid hormone, 97.9 ± 11.7 pg/ml (N < 200). Each patient received a parathyroid hormone challenge with 200 units of PTH and the absolute change in per cent tubular reabsorption of phosphate measured. Patients' response was −10.5 ± 4.0%; normal controls, −6.7 ± 1.4% (difference not significant). There was no correlation between serum phenytoin, phenobarbital, or 25-OH-D_3 levels and FCaA. Conclusion: anticonvulsant therapy can significantly impair calcium absorption in human subjects, and this may be part of the pathogenesis in the development of osteomalacia.

Life Threatening Lactic Acidosis in Status Epilepticus

H. H. Tawadros, M.D.; D. Thiagarajan, M.D. and B. B. Mukherji, M.D., Veterans Administration Medical Center, Topeka

Scanning the literature reveals only a few reports about lactic acidosis in seizure disorders. Short-lived lactic acidosis has been recently reported complicating a single episode of grand mal seizure. However, acid base disturbances in status epilepticus have not been reported. In the two cases of status epilepticus presented here, we document that severe lactic acidosis, at times life threatening, could complicate such situations. This derangement needs close monitoring and may necessitate cautious and partial correction during the critical phase of the metabolic acidosis.

The first is a middle aged male with a long history of seizure disorder. Due to lack of compliance with anticonvulsant treatment he was admitted to ICU in status epilepticus. Severe lactic acidosis was confirmed on admission. With cautious partial correction of metabolic acidosis, gradual improvement without rebound alkalosis was noted. The second is a young male who developed repeated grand mal seizures secondary to narcotic abuse. Evaluation showed severe metabolic acidosis with increased anion gap and marked lactic acidemia. Improvement of acid base status was slow during the first four hours and needed partial correction until pH was up to 7.20, after which recovery was spontaneous, apart from mild respiratory depression secondary to narcotics. Conclusions: (1) profound acid base derangement in status epilepticus is due to rapid accumulation of lactic acid; (2) despite the severe lactic acidosis in our patients, the serum potassium level did not show any elevation; and (3) monitoring of blood gases and chemistry are essential during the critical phase of acidemia; cautious and partial correction of profound metabolic acidosis (below pH 7.20) may be needed to reduce the gravity of the life threatening situation.

Cushing's Disease With Periodic Hormonogenesis: A Case Report and Brief Review

Roscoe F. Morton, M.D.; Thomas J. Luellen, M.D. and Richard B. Lies, M.D., UKSM-Wichita

Cushing's disease is hypercortisolism due to excess pituitary production of adrenocorticotropic hormone (ACTH), and the majority of patients respond to large doses of dexamethasone (8 mg/day) by a decline in adrenal secretion of glucocorticoids. We report the case of a 40-year-old female with hypercortisolism who had a paradoxical increase in the excretion of 17-hydroxycorticosteroids and 17-ketogenic steroids during the dexamethasone suppression test. Careful study with serial determinations of the 24-hour urine excretion of 17-hydroxycorticosteroids, 17-ketogenics, and 17-ketosteroids, serum cortisol, and serum ACTH established a periodic hormonogenesis that was associated with elevated ACTH. Further evaluation has revealed no evidence of an ectopic ACTH syndrome. Despite normal polytomograms, computerized tomograms and arteriograms of the sella turcica, we postulate a pituitary microadenoma as the source of the ACTH secretion. Periodic hormonogenesis in Cushing's disease is rare, and it was first recognized in 1973.

GASTROENTEROLOGY

Acute Esophageal Obstruction: Endoscopic Management of Foreign Body or Food Bolus Impaction

William A. Hartong, M.D. and Gregory G. Rick, M.D., Shawnee Mission

Acute obstruction of the esophagus is a gastrointestinal emergency. Management depends on the cause of the obstruction as well as the predisposing abnormality.

The records of 25 patients with acute esophageal obstruction secondary to meat impaction or foreign body were reviewed to assess the effectiveness of fiberoptic management. The patients ranged in age from 4 to 69 years.

Foreign body ingestion was usually by children and accidental. Two adults ingested foreign bodies embedded in food. One sustained an esophageal perforation and mediastinitis. The other patient experienced odynophagia for 30 days before endoscopic removal of an impacted piece of plastic.

Food bolus impaction occurred most often in older patients with a predisposing esophageal abnormality. These abnormalities included: (1) esophageal stricture, (2) Schatzki ring, and (3) carcinoma of the esophagus.

All patients were managed with fiberoptic esophagoscopy. No complications were related to the endoscopy. Fiberoptic esophagoscopy is a quick, safe, and effective mode of therapy for acute esophageal obstruction.

Primary Ulceration of the Ileum Presenting as Massive Gastrointestinal Hemorrhage

G. D. Schnose, M.D. and M. J. Randles, M.D., UKSM-Wichita and St. Francis Hospital, Wichita

Single discrete ulceration of the proximal to mid ileum, unlike duodenal ulcer, is an unusual occurrence. Ulceration of the distal small bowel may be associated with protein-losing enteropathy but is only rarely complicated by massive gastrointestinal bleeding.

The case we report is of a 52-year-old white female previously in good health, who presented with a brief history of melanotic stools and a hemoglobin of 5.2 gms. After blood and volume replacement, esophagogastroduodenoscopy and sigmoidoscopy were performed. These examinations failed to identify a site of hemorrhage. As the patient continued to pass numerous bloody stools, surgical intervention was elected. The only significant finding at laparotomy was a perforated 1½ cm ulcer of the proximal-mid ileum. A segment of bowel containing the ulcer was resected and an end-to-end anastomosis performed.

Our patient is an example of primary nonspecific small bowel ulceration, a rare disease, but one that must be considered if the usual causes of upper gastrointestinal hemorrhage have been excluded. A review of this disease and of other causes of ileal ulceration is presented.

Percutaneous Transhepatic Cholangiography: Experience in a Community Hospital

Gerald Pees, M.D. and Richard M. Skibba, M.D., UKSM-Wichita

Percutaneous transhepatic cholangiography (PTC) with the "skinny" Chiba needle was performed in nine patients with cholestatic jaundice and one patient with "biliary colic." A right flank, intercostal approach resulted in bile duct opacification in all ten patients. Extrahepatic obstruction was found in eight (80%) and normal ducts in two (20%) of patients. A single, nonfatal complication (bacteremia) occurred in a patient with extrahepatic obstruction. All patients with biliary tract obstruction were operated on within 24 hours. PTC has proved to be safe and reliable in the preoperative identification of the location and nature of bile duct obstruction. It also prevented unnecessary laparotomy in patients with normal ducts. PTC is recommended as the procedure of choice for direct biliary tract opacification.

Experiences in Cimetidine Use in Upper Gastrointestinal Hemorrhage

G. L. Monto, M.D. and N. Wongsurawat, M.D., Veterans Administration Medical Center, Topeka

Cimetidine, a potent H_2-receptor antagonist, has been recently introduced for the suppression of gastric acid secretion. We conducted a retrospective and prospective study of 27 cases to see if the agent was useful in the treatment of ulceroerosive gastrointestinal hemorrhage, as it has been recommended for this purpose on theoretical grounds. Cases of duodenal ulcer, hemorrhagic gastritis, and erosive esophagitis were studied. Fourteen consecutive cases were treated by antacids or nasogastric suction only. Thirteen cases were treated with intravenous cimetidine after its introduction to this hospital in May 1978. Mean ages in the two groups were 64 (controls) and 49 (cimetidine treated). There were five ulcers and six gastritis patients among controls; ten duodenal ulcers and two gastritis patients were in the cimetidine treated group. Esophagitis and miscellaneous conditions comprised the remainder of the cases. Bleeding sites were determined — usually by x-ray and endoscopy, except in clear-cut duodenal ulcers found on x-ray or occasionally at exploratory surgery. Bleeding duration was determined by absence of occult blood in the stool. One death occurred in the control group and two in the cimetidine-treated group. Controls bled on an average of 5.6 days; cimetidine-treated patients bled 5.1 days, a difference not statistically significant ($p > 0.05$). Four patients began bleeding while on oral cimetidine. It is concluded that cimetidine does not materially affect duration of bleeding in ulceroerosive gastrointestinal hemorrhage in this population.

GENETICS

Mosaic Turner's Syndrome with Menstruation and Good Ovarian Function

R. Ranjini, M.D., Veterans Administration Medical Center, Topeka

A 27-year-old female who had been in good health was treated for depression and delusions for five years. Her menarche was normal, and she was regularly menstruating. She was of normal height with only a few minor congenital malformations. She was plump, of female phenotype with well developed external genitalia, secondary sex characteristics and uterus; ovarian function was normal. Buccal smear was chromosome positive in 55 per cent of cells, and chromosomal analysis showed mosaic XO/XX.

In 1938 Turner described a syndrome with cardinal features of short stature, sexual infantilism with primary amenorrhea, webbing of the neck, and cubitus valgus. In 1959, chromosome constitution was shown to be XO. Review of menstruation in Turner's syndrome shows that it is rare for the diagnosis to be made except when there is primary amenorrhea. Mosaicism has a diluting effect on the clinical abnormalities. There is a wide spectrum of clinical and somatic abnormalities with varying height, without webbing of the neck, diversity in somatic malformations, with poor ovarian function to well developed ovaries, and even pregnancy has been reported. Mosaicism is said to be present when the different cell lines are present simultaneously in all or some of the tissues. In mosaics, chromosome examination of two or more tissues is mandatory, as degree of admixture of component stem lines may vary widely between tissues. Mosaicism follows an error in mitosis during an early cleavage division after conception with inclusion of both cell lines in one, or it may arise through anaphase lagging.

HEART, CIRCULATION

The Effect of Concurrent Oral Disopyramide and Propranolol on Cardiac Function in Healthy Men

James Coker, M.D.; William Cathcart-Rake, M.D. and Daniel Azarnoff, M.D., UKSM-KC

Ten healthy men were evaluated for left ventricular (LV) performance changes and β-blockade caused by oral propranolol (P) and disopyramide (D). LV function was assessed by echocardiographically determined ejection fraction (EF) and measurement of systolic time intervals. β-blockade was assessed by three min exercise heart rate. In five volunteers (group I) these tests were performed before and following the AM dose of drug(s) at the end of four consecutive study periods: single dose P (80 mg); one week D (200 mg q 6 hrs); one week D + single dose P, one week P (80 mg q 8 hrs), and D. The other five (group II) were studied after: single dose D (200 mg); one week P; one week P + single dose D; one week P, and D. Single dose P caused a slight but insignificant decrease in EF and increase in pre-ejection period index (PEPI). In contrast, single dose D decreased EF (mean ± SEM) by 15 ± 2% ($p < 0.005$) and increased PEPI by 16 ± 2 msec ($p < 0.005$). One week P caused minimal changes in EF and PEPI, while one week D decreased EF by 10 ± 4% ($p < 0.05$) and increased PEPI by 16 ± 7 msec ($p < 0.05$). There were no additive effects of combined P and D. In group I, EF and PEPI returned toward normal during the final study period, suggesting that tolerance to the negative inotropic

effects of D may occur. Finally, we confirmed that D lacks β-antagonistic activity and does not influence β-blockade by P.

Radiation Pericarditis — Late Onset

R. David Holmsten, M.D. and B. B. Mukherji, M.D., Veterans Administration Medical Center, Topeka

A 57-year-old male was admitted because of shortness of breath and orthopnea. Twenty years earlier he had received irradiation to the chest for metastatic embryonal cell carcinoma of the testes. Significant physical findings included: distended neck veins, grade III/VI pansystolic murmur at the apex, enlarged tender liver, and 3+ pitting edema of the ankles. There were old radiation burns of the anterior and posterior chest wall. Chest x-ray revealed pleural effusions with an enlarged silhouette. An echocardiogram revealed an enlarged LV with a thickened ventricular wall and pleural effusions. The patient was managed with diuresis and thoracentesis with prompt recurrence of the pleural effusions. Cardiac catheterization revealed CI of 1.7 L/M^2, SV of 28 ml. Pressures were: RA μ = 6, RV 65/6, PA 65/26, P wedge μ = 24, LV 112/16, aorta 112/62. Ejection fraction was 59%, estimated regurgitant volume was 74%. The impression was severe mitral regurgitation. At surgery, extensive fibrosis of the pericardium and mediastinum was noted, but no pericardial fluid. A porcine mitral valve was inserted. Following surgery the patient did not improve. One month later a Swan-Ganz catheterization demonstrated findings consistent with constrictive pericarditis. He was returned to surgery but no pericardial fluid was found. He continued to deteriorate and died seven days later. No evidence of carcinoma or coronary artery disease was found at autopsy. There was extensive mediastinal and pericardial fibrosis. The myocardium was spared of any significant fibrosis. Conclusion: clinical constrictive pericarditis may appear as long as 20 years post radiation, although this is not the usual presentation.

Echocardiographic Findings Predicting the Success of Pericardiocentesis

Jay W. Murphy, M.D.; Bert Wong, M.D. and Khatab Hassanein, Ph.D., UKSM-KC

Pericardiocentesis (PC) is an effective method of obtaining pericardial fluid, but it can result in serious complications. To determine the ability of the echocardiogram (ECHO) to select patients for successful and uncomplicated PC, we compared the ECHO done immediately prior to PC with the results of PC. Twenty patients (Group I) had a successful PC. Ten patients (Group II) had an unsuccessful or complicated PC (seven — no fluid obtained; two — cardiac tamponade not relieved; one — right ventricle punctured). The mean ECHO measurement of the anterior pericardial clear space (APCS) was smaller in patients having an unsuccessful or complicated PC (Group I — 20.65 ± 2.35 mm; Group II — 8.10 ± 1.77 mm; $P < 0.005$). Of 20 patients with an APCS > 10 mm, 17 (85%) had a successful PC. Of ten patients with an APCS < 10 mm, seven (70%) had an unsuccessful or complicated PC. There was no statistically significant difference between the two groups in the mean ECHO measurements of the right ventricular, left ventricular, total heart or total pericardial dimensions, the ECHO estimate of the volume of pericardial effusion, or the size of the posterior pericardial clear space. The demonstration of a small (< 10 mm) APCS identifies patients in whom pericardiocentesis is likely to be unsuccessful or complicated.

Constrictive Pericarditis Following Acute Idiopathic Pericarditis

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A definite syndrome of pericardial constriction has been reported following acute viral or idiopathic pericarditis in young patients. Constrictive pericarditis continues to be an uncommon entity associated with a rather difficult differential diagnosis. We report a case of pericardial constriction occurring in a 23-year-old caucasian male six weeks post acute pericarditis. This patient presented with dyspnea and ascites as the syndrome developed. In addition he displayed marked jugular venous distension with typical right heart pressures, hepatomegaly, and poor response to medical treatment with digitalis and diuretics. Surgical pericardiectomy provided relief of symptoms and gradual resolution of constriction. In young and middle-aged patients with signs and symptoms of right heart failure, pericardial constriction must always be considered.

Development of Pericardial Tamponade in a Patient with Multiple Myeloma

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Multiple myeloma is a disease with many protean manifestations. Painful bony lesions and fractures have been considered the hallmark of multiple myeloma. Soft tissue plasmacytomas have been seen in rare patients. A 54-year-old woman with a two-year history of multiple myeloma presented with a one-month history of shoulder pain, cough, and dyspnea on exertion progressing to dyspnea at rest. The patient was found to have a pericardial effusion. Pericardiocentesis and subsequent surgical drainage was performed. Pericardial fluid contained malignant plasma cells. Biopsy showed involvement of the pericardium and myocardium with malignant plasma cells. The patient continued to have clinical signs of pericardial tamponade. The patient was taken to surgery and found to have involvement of the entire pericardial lining with myeloma. She died the following day. An autopsy was obtained. Management and a review of the literature are presented.

Left Ventricular Function and Late Mortality Following Coronary Artery Bypass in Patients with Severe Left Ventricular Dysfunction

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The operative and late courses of 90 consecutive patients (pts) (mean age = 54 yrs) with poor left ventricular function (LVF) who had coronary artery bypass (CABP) during 1971-75 were analyzed. Preoperative LV ejection fractions (EF) ranged from 16-40 per cent (mean = 32%). Sixty-five pts had 3-vessel coronary artery disease (CAD) (14 with main left CAD), 21 pts had 2-vessel CAD, and four pts had 1-vessel CAD. There were three (3.3%) operative deaths and four (4.4%) perioperative myocardial infarctions (MI) (2 fatal). Seventy-five pts had class III-IV angina preoperatively; 53 (71%) were completely relieved of angina, and 13 (17%) partially relieved at the end of a mean followup period of 40.5 months. There were nine (10%) cardiac or unexplained late deaths, and 11 (12%) nonfatal late MIs. Thirty-one pts underwent catheterization at a mean of 19 mos postoperatively. LV EF had increased ≥ 10% in 13 (42%), was unchanged in 15 (48%), and had decreased > 10% in three (10%).

These data would indicate that pts with poor LVF can undergo CABP at an acceptable risk (3.3%) with combined operative and late mortality and nonfatal MI rates of 3.6 per cent/yr and 3.8 per cent/yr, respectively. The operative mortality of CABP is greater in pts with poor LVF than in our overall experience (3.3% vs 0.8%). However, these late results suggest improved survival, frequent relief of symptoms, and preservation or im-

provement in LV function in this higher risk subset of patients with CAD.

"Sham Operation" Revisited: A Comparison of Complete vs Unsuccessful Coronary Artery Bypass

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Of 1795 consecutive pts who had coronary bypass surgery (CBPS) between 1971-75, 446 had subsequent bypass graft (BP) visualization and coronary arteriography. Of these, 56 pts had no patent BP (Group I); 54 pts had complete revascularization (revasc) with patent grafts bypassing all coronary disease (CAD) (Group II).

Pre-OP	*FC I-II*	*FC III-IV*	*NLV*	*SLV*	*SVD*	*TVD*
Group I (56 pts)	12	44	15	20	17	22
Group II (54 pts)	5	49	25	19	35	5
*Post-OP**	*FC I-II*	*FC III-IV*	*NLV*	*SLV*	*MI*	*Death*
Group I	41	15	7	40	24	5
Group II	49	5	27	18	8	3

* Mean followup = 47 mos; NLV = normal left ventricular function, SLV = severe LV dysfunction, SVD and TVD = 1- and 3-vessel CAD, respectively, MI = myocardial infarction.

We conclude that (1) CBPS pts experience impressive symptomatic improvement regardless of the completeness of revasc; (2) global LV function deteriorates with unsuccessful surgery but is generally unchanged with complete revasc; (3) pts with complete revasc have fewer MIs (4%/yr vs 11%/yr) and deaths (1%/yr vs 2%/yr) but are not returned to a "general population" risk of coronary events; and (4) the late survival of unsuccessfully revasc pts is more favorable than predicted from previous natural history data. The latter suggests that factors other than CBPS may play a role in the long-term survival of CAD pts undergoing surgery.

Heart Surgery in Topeka — Nine Hundred Cases

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Analysis of the first 900 patients undergoing heart surgery in Topeka is presented. Surgical indications were coronary artery disease, valvular heart disease, complications of myocardial infarction (aneurism, recurrent ischemic symptoms, mitral regurgitation, septal defect, or intractable arrhythmia), and congenital heart disease. Operations were performed with cardiopulmonary bypass, hemodilution, hypothermia, and cardioplegic infusion during aortic cross clamping.

The most frequent surgical procedure was aorto-coronary bypass for triple vessel disease, averaging 3.5 grafts/patient, with an early mortality in uncomplicated cases of less than 1 per cent. Early and late results, as well as management of complications, will be presented. The management of complex problems — such as bypass and aneurismectomy, bypass and valvular surgery, and surgery in the presence of diminished ventricular function — will be discussed.

It is evident on the basis of the data, that a smoothly functioning team, adequate case volume, and proper support, can assure excellent results for cardiac surgical patients in a community hospital.

Recurrent Pattern of Congestive Heart Failure in Alcoholic Cardiomyopathy

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Documented here is the clinical course of a patient with alcoholic cardiomyopathy (AC) in whom repeated episodes of congestive heart failure (CHF) over a period of ten years precipitated by alcoholic intake were eventually controlled by total abstinence.

A 48-year-old male veteran who admitted to chronic ethanol abuse presented to us in 1975 with atrial fibrillation and severe CHF following an alcoholic binge. He gave a history of recurrent episodes of atrial fibrillation with CHF dating from 1968. There was no history of rheumatic fever, diabetes mellitus or ischemic heart disease. He responded promptly to digitalis and diuretic therapy. Cardiac catheterization data were consistent with congestive cardiomyopathy; coronary angiography was normal. Prolonged bed rest was not instituted, and the patient was discharged on digitalis, diuretics, and coumadin. Between 1975 and 1977, he was readmitted seven times with CHF precipitated by alcohol abuse. Each episode responded promptly to continuation of digitalis, diuretics, and cessation of alcohol intake without any prolonged bed rest. In March 1977, he developed retroperitoneal hemorrhage with massive bleeding into his right leg as a result of coumadin-alcohol interaction. Following this event, with much persuasion, the patient abstained from ethanol intake. During the subsequent 18 months of observation, the patient had no further episodes of CHF. Currently, he is asymptomatic and fully employed. Conclusion: (1) total abstinence may lead to clinical reversal of AC even in long-standing cases; (2) prolonged bed rest is neither indicated nor practicable in AC; and (3) routine prophylactic anticoagulation as an outpatient could be hazardous in this group of patients.

Simultaneous Occurrence of Coarctation of the Aorta and Asymmetric Septal Hypertrophy in an Adult Patient

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Coarctation of the aorta is a disorder of unknown cause that comprises 3-10 per cent of all congenital heart diseases. Asymmetric septal hypertrophy (ASH) is an inherited disorder that is thought to be transmitted as an autosomal dominant trait, although many cases are not clinically apparent. Although the occurrence of these two disorders in the same patient has been described before, it is quite uncommon and should be recognized before an attempt is made to correct the coarctation in order to avoid exacerbation of symptoms due to the ASH.

We describe a 22-year-old white male who presented for evaluation of hypertension. Examination of the chest, heart, and extremities revealed findings compatible with the diagnosis of coarctation of the aorta; chest x-ray was confirmatory. In addition the patient had a systolic murmur which was characteristic of that heard in patients with ASH. Echocardiogram revealed marked asymmetric septal hypertrophy. One year after surgical correction of the coarctation the patient is doing well on a modest dose of propranolol.

The clinical manifestations of these two cardiac lesions are reviewed and the effect of the coarctation on the abnormal physiology induced by ASH is described.

Cardiomyopathy in Diabetes Mellitus

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Cardiomyopathy is a disease primarily involving the myocardium excluding ischemic heart disease, hypertensive cardiovascular disease, cor pulmonale, valvular heart disease, and congenital heart disease. Diabetes mellitus is known to cause angiopathy in addition to atherosclerotic heart disease.

We studied all cases of cardiomyopathy at Halstead Hospital 1975-1977 to determine the incidence of cardiomyopathy in diabetes mellitus without evidence of coronary atherosclerotic heart disease.

There were 18 patients with the diagnosis of cardiomyopathy. Three patients had a history of diabetes mellitus; a fourth had

reactive hypoglycemia with high insulin output — possibly a prediabetic. Four patients out of 18 is a significant number to be just incidental. We noted that these four patients had no history of angina, high blood pressure, congenital heart disease, or valvular heart disease. Two of them had normal coronary arteriograms.

Cardiomyopathy in diabetes mellitus is due to small vessel disease as a result of angiopathy.

Changes in R Wave Amplitude With Treadmill Stress Testing

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ST segment changes with treadmill exercise testing may be misleading. This study was done to determine if changes in R wave amplitude would improve the sensitivity and/or specificity of such tests. Treadmill exercise tests and coronary angiograms were compared in 62 randomly selected patients (32 females, 30 males). Changes in R wave amplitude with exercise were compared to ST segment changes. An increase or no change in the R wave in lead V5 was taken as an abnormal response (Brody effect), while a decrease in the R wave amplitude was a normal response. Horizontal or downsloping ST segment depression of $\geq$ 1 mm, 80 msec from the J point was defined as an abnormal ST segment response. Comparison of R wave and ST segment sensitivity and specificity were as follows:

	Sensitivity (%)		*Specificity (%)*	
	ST	*R*	*ST*	*R*
All patients	83	78	79	34
Males	91	91	75	42
Females	66	50	81	31

We conclude that exercise-induced changes in R wave amplitude are no more sensitive and are less specific than conventional ST segment changes.

Does Postoperative Arteriography Predict the Late Course Following Coronary Bypass Surgery?

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The courses of 446 consecutive patients (pts) surviving coronary artery bypass surgery (CABS) who had postoperative (po) coronary and bypass graft angiography were related to po LV function and the distribution of "residual" coronary disease (CAD) (*i.e.* $\geq$ 70% occlusion not bypassed by a patent SVG). Fifty-four pts were completely revascularized with all CAD bypassed by patent SVGs (Group 0); 39 pts had "residual" single-vessel left anterior descending disease (Group Ia), 50 right coronary artery disease (Group Ib), and 60 left circumflex disease (Group Ic); 172 2-vessel CAD (Group II); and 71 3-vessel CAD (Group III). The results of followup over a mean period of 50 months were:

	Asymptomatic (%)	*Myocardial Infarct (%/yr)*	*Mortality (%/yr)*
Group 0	86	3.5	1.8
Group Ia,Ib,Ic	89, 78, 77	3.7, 3.4, 4.8	0.6, 0.5, 1.2
Group II	70	2.4	1.4
Group III	78	8.1	3.4

Except for "residual" 3-vessel disease, the late po CABS course could not be predicted from the extent of "residual" CAD or po LV function. The po course in each subgroup of "residual" CAD was much more favorable than predicted from previous natural history studies, emphasizing the difficulty in attributing improved po survival to CABS alone.

HEMATOLOGY

Nafcillin-Associated Granulocytopenia

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Granulocytopenia has been infrequently associated with the therapeutic use of the penicillin antibiotics. Previously only five cases of nafcillin associated granulocytopenia have been reported, but we have observed three such patients during a 12-month period. *Patient one* had *S. aureus* endocarditis. There was appropriate initial response with defervescence and negative blood cultures. By day 21, there was leukopenia with only 1500 granulocytes. Nafcillin was stopped and cure was achieved with cefazolin for a total of six weeks of therapy. *Patient two* was a 15-year-old with hematogenous osteomyelitis of the right femur caused by *S. aureus*. On day 17, leukopenia was observed decreasing to 250 granulocytes on day 19. Cefazolin was substituted and cure was achieved after seven more days. *Patient three,* who required chronic hemodialysis, developed access site infection. Granulocytopenia occurred on day 22 of nafcillin therapy. Vancomycin resulted in cure. Spontaneous recovery was observed after cessation of nafcillin therapy in these patients. Examination of bone marrow from two of the three patients showed maturation arrest and eosinophilia. An immune meditated mechanism was sought but not found. Thus, we suspect nafcillin or a metabolite may exert an inhibitory effect on granulocyte formation by the bone marrow. Patients who are being treated with nafcillin for longer than two weeks should have twice weekly leukocyte and differential counts. If granulocytopenia is observed, nafcillin should be stopped and therapy reinstituted with a cephalosporin or vancomycin.

Lithium Carbonate Therapy for Granulocytopenia in a Patient With Myelofibrosis

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The administration of lithium carbonate has been shown to induce granulocytosis in patients with psychiatric disorders, Felty's syndrome, and attenuate the granulocytopenia associated with cancer chemotherapy. In a patient with documented myelofibrosis (bone marrow biopsy) with associated granulocytopenia, lithium carbonate 300 mg 3 times/day orally, with appropriate serum levels of lithium documented, was begun in hopes of raising the granulocyte level. Results are shown below.

Time	*White Blood Cell Count (Total)*	*Neutrophils*	*Lympho-cytes*	*Mono-cytes*
Zero	1900	50% (total = 950)	49%	1%
One week	2400	50% (total = 1200)	32%	11%
Two weeks	2900	60% (total = 1740)	22%	14%

It is concluded from this one patient with myelofibrosis that lithium carbonate did aid in inducing a higher circulating level of granulocytes. It was not shown from this case whether the increased granulocyte level was secondary to demargination of granulocytes, increased production, or a longer survival time.

INFECTIOUS DISEASES

Rocky Mountain Spotted Fever (RMSF) Seen in Wichita

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Four cases of RMSF seen in Wichita in the past two years are reviewed. No case was diagnosed prior to the development of the classical skin rash and, in three of the four cases, encephalopathy with coma occurred after hospitalization for three to seven days with fever and rash. Patients A and B were initially treated with inappropriate antimicrobials (ampicillin and cephalexin, respectively). Patient C had a concurrent enteroviral infection of the central nervous system which delayed the diagnosis of RMSF. All patients improved rapidly upon administration of appropriate antimicrobials (chloramphenicol or tetracycline), and none had permanent sequelae. Only patient B had a known tick bite; however, both patients A and D had appropriate environmental exposure. The presumed incubation time ranged from four to nine days. Serological conversion eventually occurred in all patients, but was initially negative and misleading in two cases. A clinical summary is shown below:

				Peak		*Interval between onset and:*	
Pt	*Age*	*Sex*	*Incub Time*	*OX19 titer*	*Coma*	*Rash*	*Treatment*
A	21	F	9 d	1:1280	yes	3 d	9 d
B	23	F	4 d	1:4000	yes	8 d	13 d
C	4	M	?	1:640	yes	3 d	9 d
D	46	M	8 d	1:40	no	6 d	7 d

The early diagnosis and proper therapy of RMSF is dependent upon clinical suspicion rather than laboratory findings. RMSF must be considered in patients with a centripetally distributed febrile rash associated with encephalopathy.

Severe Myositis During Simultaneous Infection With β-Hemolytic Streptococcus and a Viral Agent: A Possible Potentiating Effect

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Synergistic or potentiating effects between viral and bacterial agents have been documented in laboratory animals, but have not been well established clinically. We report two patients seen during a two-week period with simultaneous viral and bacterial infection whose fulminating courses indicate that enhanced virulence may have occurred.

Case One: A 28-year-old woman presented with generalized myalgia, arthralgia, knee effusion, and oliguria. CPK reached 152,000 IU/l, acute renal failure developed, and hemodialysis was begun after creatinine rose beyond 10 mg/dl. Group A β-hemolytic streptococcus grew from knee and blood. Convalescent titers of Coxsackie B-2, B-3, B-4, and B-6 all showed a four-fold rise, but viral cultures were negative. Muscle biopsy showed focal necrosis with regeneration.

Case Two: A 67-year-old man was admitted with fever, chills, myalgia, a CPK of 33,700 IU/l, myoglobinemia and myoglobinuria. Disseminated intravascular coagulation rapidly developed and death occurred five hours after admission. Blood, spinal fluid, and skin cultures grew Group A β-hemolytic streptococcus. Electron microscopy of skeletal muscle showed infiltration of fascicles by a picorna virus. Viral cultures of blood and feces were negative.

This evidence for simultaneous viral and bacterial infection with rhabdomyolysis, and fulminant clinical course suggests that enhancement of infection may occur during streptococcal and viral infections. Enhanced effects of combined infections with streptococcus and virus have not been recognized previously in man. Further studies may elucidate this relationship.

Kawasaki Disease — Five Cases in Young Adults

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Kawasaki disease or mucocutaneous lymph node syndrome was first described in 1967 as an acute febrile illness of unknown cause. The characteristic syndrome includes fever, conjunctivitis, erythema of the oropharynx, erythema of the palms and soles, followed by desquamation, truncal exanthem, and cervical lymphadenopathy. All patients previously reported have been children.

In the past two years we have observed five young adults with Kawasaki disease: four females and one male, ages 19-28 yrs. All presented with an acute febrile illness and the majority of the features listed above. Four patients had laboratory evidence of renal dysfunction, four had hypotensive episodes, and three had clinical features of myocarditis. All patients recovered fully from the acute illness. However, one patient died suddenly 11 mos following his acute illness and coronary arteritis was determined at autopsy.

The pathophysiology appears to be an acute microvascular endothelial and diffuse epithelial injury to skin and mucous membranes. Although a mortality of only 1-2 per cent has been reported in children, this illness was very severe in these adults. Our experience would suggest high dose steroids may be of benefit to these patients.

Pseudomycosis Caused by Fungal Contaminant in Histological Stain

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Silver stains of tissue specimens are important in the evaluation of patients with suspected fungal infections. In certain instances, fungal morphology on examination of these specimens is distinct enough to permit presumptive diagnosis and subsequent therapy. Recently, we cared for a patient who had nodular lesions of the lower extremities. Initial biopsy of one lesion showed noncaseating granuloma formation on microscopic examination. Grocott methenamine silver stained sections showed budding yeast. Because fungal cultures were not obtained, a second biopsy with cultures and histologic examination was done. Cultures for bacteria, mycobacteria, and fungi revealed no growth. Histologic examination again revealed noncaseating granulomata but no budding yeast on silver stained sections. A third biopsy confirmed the observations of the second biopsy. A second patient with steroid dependent chronic obstructive lung disease was evaluated for *Pneumocystis carinii* pneumonia with Grocott methenamine silver stain on expectorated sputum. Examination of this preparation showed budding yeast. The patient recovered without specific therapy and cultures of the sputum for fungi were negative. Examination of the light green counterstain used in the silver staining procedure showed budding yeast similar to that seen in the specimens from the two patients. Culture of the counterstain grew *Phialophora jeanselmei*. Because fungal organisms may contaminate and grow in histological stains such as light green, both the primary physician and pathologist must be aware of this as a cause of pseudomycosis. Ways of preventing this problem will be discussed.

Fatal Disseminated Intercellular Infection in a Patient With Thymoma

Garold Minns, M.D.; Roy Weber, M.D.; Douglas W. Voth, M.D. and H. C. Goodpasture, M.D., UKSM-Wichita

Disorders of the thymus gland may lead to diminished host defenses but tumors of the thymus have not been commonly

associated with fulminant disseminated intercellular infection. We report a 74-year-old female who experienced fever, malaise, weight loss, and hepatic dysfunction for one year and a cystic right pulmonary mass which was known to be present for 25 years. A liver biopsy revealed granulomatous hepatitis, but special stains, extensive cultures, numerous antibody and protein measurements, and observation of delayed type hypersensitivity were non-diagnostic. Sulfa hypersensitivity was suspected, and when signs and symptoms of the illness progressed, corticosteroids were administered. *Histoplasma capsulatum* were seen on Wright stains of the patient's peripheral blood and amphotericin B was administered; but the patient rapidly deteriorated and died in pulmonary, hepatic, and renal failure. The autopsy findings showed evidence of disseminated infection with both histoplasma and cytomegalovirus as well as an encapsulated necrotic thymoma.

This patient demonstrates several potential relationships between tumors of the thymus gland and microbial agents that cause intercellular infection.

KIDNEY, ELECTROLYTES & HYPERTENSION

A New Hemodialysis Technique for Treatment of Refractory Heart Failure

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Isolated ultrafiltration (UF) is a technique that has recently been developed in hemodialysis. UF facilitates removal of large volumes of fluid from patients in a short period of time without causing significant hypotension which would usually occur in this setting with conventional hemodialysis. The negligible cardiovascular effect with this procedure has been attributed to either a lack of significant plasma osmolality change or the omission of contact with acetate, which is normally present in the dialysate of conventional hemodialysis. These factors allow normal blood pressure regulatory processes to remain operative and minimize myocardial depressant activity respectively.

A patient with refractory biventricular heart failure is presented in whom serious hypersensitivity to all forms of diuretic therapy precluded conservative management. Renal function was moderately impaired due to diabetic nephropathy; however, not to the degree that maintenance dialysis would have been required. UF was accomplished for two hours three times/wk, and an average of 3.4 kg fluid weight was removed/treatment session. No significant hypotensive episodes occurred during the treatment periods, in spite of the large volume of fluid removed over a relatively short period of time.

UF seems to offer a promising new therapeutic regimen for refractory heart failure with advantages of immediate and predictable response, avoidance of acid-base disturbances inherent with potent diuretic therapy, and the potential for reducing time of hospital stays and overall expense of treatment.

Rapidly Progressive Glomerulonephritis: A Favorable Response to Pulse Corticosteroid Therapy

Michael Saunders, M.D. and Dennis Ross, M.D., UKSM-Wichita

Idiopathic RPGN is a severe form of renal disease characterized by abrupt onset, rapid decline in renal function, and poor outlook for recovery. Therapeutic modalities have been disappointing.

We have recently seen a patient with idiopathic RPGN who presented with a creatinine of 11.4 mg/100 ml, BUN of 190 mg/100 ml, and oliguria. Renal biopsy was performed on the third hospital day after starting the patient on hemodialysis and excluding other causes of renal failure. The biopsy revealed extensive crescentic formation, subepithelial "humps," exudation, and intracapillary deposits. The patient was given 1 gm/day methylprednisolone intravenously for five days followed by a tapering dose of oral prednisone therapy. She required intermittent dialytic therapy for three weeks, but recovered sufficient renal function to discontinue hemodialysis. Three consecutive creatinine clearances were 18 ml/min.

This report suggests the possibility of recovery of renal function in severe idiopathic RPGN with pulse dose steroid treatment and points out the necessity of early diagnosis and therapy.

Severe Hyperchloremic Acidosis Complicating Jejunoileal Bypass

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The intestinal bypass operation for treating morbid obesity may result in serious complications such as liver damage, renal failure, bypass enteropathy, and malnutrition. Hyperchloremic acidosis is a rare complication and, if present, tends to occur quite early after the operation. We present a case of late hyperchloremic acidosis complicating jejunoileal bypass.

A 45-year-old male was admitted with severe general weakness. He had been obese since childhood. He had an aunt who died at age 70 with a weight of 600 lbs. Ten years prior to this admission the patient underwent jejunoileal bypass. He weighed 480 lbs at that time. There was no significant loss of weight after surgery. On this admission, the patient weighed 550 lbs. He had experienced increasing symptoms of diarrhea up to 10 times/day during the past three months. There was severe muscle weakness, hyporeflexia, and mental obtundation. The serum electrolytes showed: Na, 132 mEq/l; K, 3.2 mEq/l; CO_2, 15 mEq/l; and Cl, 111 mEq/l. Arterial blood gases revealed mixed respiratory alkalosis and metabolic acidosis: pH, 7.47; pCO_2, 17.3; and CO_2, 12.6. Serum ammonia was 98 micromoles/l; serum creatinine, 1.9 mg/dl; BUN, 10 mg/dl. The stool pH was 7.9. Urine pH was 5, and no glucose or protein was found. Oral and intravenous sodium bicarbonate, protein restriction, and other supportive measures failed to improve the patient's rapidly deteriorating condition. Conclusion: (1) severe hyperchloremic acidosis may develop as a late complication of jejunoileal bypass and is due to intestinal loss of bicarbonate; renal tubular acidosis is unlikely; and (2) patients may continue to gain weight after shunt surgery.

Renal Failure Secondary to Diffuse Neoplastic Involvement of the Kidneys

Brad Stuewe, M.D. and Arnold Chonko, M.D., UKSM-KC

Acute renal failure (ARF) may occur for several reasons in patients with nonrenal malignancies. Urinary tract obstruction due to tumor mass or retroperitoneal fibrosis, hypercalcemia, hyperuricemia, and nephrotoxic antibiotics are more common causes of ARF in patients with malignancy. Nephrotoxicity due to chemotherapeutic drugs and secondary amyloidosis are less frequently seen. Direct tumor involvement of the kidneys is a rare cause of ARF; when it occurs it is generally thought to portend a grave prognosis. We report two interesting cases of ARF.

Case One: A 67-yr-old white male with previously documented lymphoblastic lymphoma presented with oliguric ARF. Diffuse infiltration of his kidneys without ureteral obstruction was suggested by findings of abdominal sonography and CAT scan. Malignant cells were clearly present in the urine sediment. The oliguria (< 30 ml/day) and azotemia (BUN, 125; Cr, 11 mg/dl) on admission cleared rapidly with the institution of radiation therapy.

Case Two: A 45-yr-old white male presented with left knee pain and angina pectoris. Biopsy of the left tibia revealed histiocytic lymphoma. Shortly thereafter progressive oliguria developed (< 50 ml/day) and his serum creatinine rose to 12 mg/dl. Retrograde pyelograms revealed enlarged kidneys with-

out evidence of obstruction. A renal biopsy revealed diffuse infiltration of the renal parenchyma with malignant appearing histiocytes. Chemotherapy was begun. Urine output improved stepwise to 2-3 liters daily over eight days. Serum creatinine was 1.2 mg/dl at discharge.

Thus, ARF due to diffuse infiltrative malignancy with responsive tumors should not preclude aggressive therapy.

Pyridoxine Controlled Seizures in a Dialysis Patient

Edward Wood, M.D., Topeka

A frequent complication of renal dialysis is major motor seizures, often thought to be a manifestation of the disequilibrium syndrome and other electrolyte abnormities. The subject of this presentation, a 59-year-old female on chronic dialysis, was admitted with the acute onset of tonic-clonic seizures. Regular medications had included the following: isoniazid, ethambutol, pyridoxine, multiple vitamins, and aluminum hydroxide. In spite of aggressive and usual therapy for major motor seizures, the patient did not improve and indeed became semicomatose. Isoniazid toxicity was then considered, and accordingly she was treated with large doses of pyridoxine with resolution of neurologic symptoms and signs. It is believed that this patient represented an unusual manifestation of isoniazid toxicity — namely seizures — which apparently are responsive to pyridoxine administration.

The Effects of Gentamicin (G) on Renal Function in the Sexually Mature vs Sexually Immature Rabbit

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Gentamicin therapy is associated with a higher incidence of nephrotoxicity in adults than in children. To study the relationship of age to nephrotoxicity, G was administered 15 mg/kg/bid to 12 sexually immature (SI) and 13 mature (SM) rabbits. Age-matched controls received placebo (C). The animals were killed after 28 days or when serum creatinine (Cr) levels exceeded 3 mg/dl. Tissue was obtained for histology and G levels. Two mm segments of proximal straight tubule (PST) were incubated in rabbit serum with ^{3}H-PAH for 60 min; tissue/medium ^{3}H-PAH ratios were determined.

Group	*Serum Cr mg/dl*	*I/M ^{3}H-PAH*	*Histology*	*Cortex/Medulla G levels g/ml*
SI(C)	< 1.5	206 ± 29	normal	0/0
SI(G)	< 1.5	167 ± 30	cytosegresomes	174/59
SM(C)	< 1.6	111 ± 22	normal	0/0
SM(G)	< 2.0	80 ± 16	cytosegresomes	196/84
SM(G)	> 3.0	9 ± 2	necrosis	234/78

No SI rabbits developed renal failure; 7/13 SM rabbits developed serum Cr > 3 mg/dl. There were no major differences in serum or tissue G levels. There was tubular cytosegresome formation in all G animals. ^{3}H-PAH uptake by PST was severely depressed only when tubular necrosis was present. ^{3}H-PAH T/M levels in PST from SI animals treated seven days were elevated when compared to controls (277 ± 37 vs 143 ± 27). Thus, G given to SI animals produced an early augmentation of PAH uptake (seven days) with a later decline (28 days). The development of renal failure in consequence of tubular injury was clearly related to the maturity of the animals. The G-induced augmentation of organic acid uptake into proximal tubule cells could account for the reports of enhanced nephrotoxicity of cephalosporin and aminoglycoside antibiotics when used in combination.

NEUROLOGY

Ganglioneuroma: Unusual Tumor Presenting With Headaches

Darrell L. Ellis, M.D.; Lauren K. Welch, M.D. and Jay Gold, M.D., Wesley Medical Center, Wichita

A case report is presented of a 22-year-old white male with a ten-year history of headaches, manifested by bilateral eye pain and left visual field teichopsia. The headaches increased in severity and intensity two weeks prior to admission. Physical examination was remarkable for a left homonymous hemianopsia and mild bilateral papilledema. CT scan showed a large cystic tumor of the right parietal, posterotemporal, and occipital region with a 4 x 5 cm mass in the posterior portion containing punctate areas of calcification. Pathology of the tumor revealed a ganglioneuroma, which initially appeared to be a ganglioglioma. A review of this tumor's place in the classification of brain tumors, pathologic characteristics, course, prognosis and treatment is provided.

Carcinomatous Amyotrophic Lateral Sclerosis in a Patient With Multiple Myeloma

Scott Pace, M.D. and Lauren K. Welch, M.D., UKSM-Wichita and Wesley Medical Center, Wichita

During the last several decades, the relationship between cancer and remote neurological syndromes has been noted by many physicians. These neurological syndromes appear to be unrelated to direct invasion by tumor or to metastatic involvement. General categories of nonmetastatic neurological manifestations of malignant disease include muscular disorders, involvement of the neuromuscular junction, peripheral neuropathies, anterior horn cell and motor neuron disorders, and central nervous system syndromes such as diffuse encephalopathies. Multiple causes for these disorders have been implicated including infectious, metabolic, toxic, autoimmune, and nutritional. Possibly a common agent produces both the cancer and the neuropathology or perhaps the tumor itself produces a circulating peptide as seen in many endocrine effects of cancer.

In this report, a 63-year-old female developed amyotrophic lateral sclerosis three years after she was diagnosed as having multiple myeloma. Previously other authors have correlated amyotrophic lateral sclerosis with cancer implying possible causative significance. This patient's clinical presentation supports a strong correlation between her myeloma and her neurological syndrome.

Hypothalamic Syndromes Produced by Cerebral Subarachnoid Cyst

R. Ranjini, M.D.; Subramoney Nagaswami, M.D. and Thomas Mathews, M.D., Veterans Administration Medical Center, Topeka

Manifestation of hypothalamic syndrome due to cerebral subarachnoid cysts is a rare entity as yet unreported. It presents a diagnostic challenge. As subarachnoid cysts are amenable to surgery, it is also one of the few remediable causes. Presented are two cases seen at the Topeka Veterans Administration Medical Center.

Case One: A 50-year-old male with malignant depression of long standing presented with severe anorexia nervosa of one year's duration leading to cachexia. In spite of extensive work-up of the patient, no cause was found until CAT scan and cerebral angiography were done.

Case Two: A 39-year-old male with a history of head injury 20 years earlier, presented with headache, confusion, excessive thirst, and hunger. Investigation of polydipsia and polyuria showed an inappropriate ADH syndrome. All investigations were negative except that in the light of *Case One*, CAT scan and angiography showed the same pattern, with successful resolution of the case by surgery.

Both of these cases showed subarachnoid cyst in the Sylvian fissure, producing pressure symptoms in the hypothalamic area. *Case One* exemplifies the cause of subarachnoid cyst due to congenital cell nests during development and producing symp-

toms later with the growth of the cyst due to a ball-valve action. *Case Two* is an example of head injury pinching off a portion of arachnoid membrane to form cystic change.

PULMONARY DISEASE

The Relationship of Gastroesophageal Reflux to Reversible Airways Disease

George A. Vraney, M.D. and Charles Pokorny, M.D., The Hertzler Clinic, Halstead

The relationship of gastroesophageal reflux and reversible airways disease has been frequently postulated with the support for this relationship largely remaining anecdotal. In some studies of patients with a variety of pulmonary disorders, an increased incidence of gastroesophageal reflux has been documented. In an effort to evaluate this relationship further, 100 patients with documented gastroesophageal reflux on upper gastrointestinal radiographic evaluation were evaluated for changes in pulmonary function compared with 100 age and sex matched patients demonstrating no spontaneous reflux on the same day as evaluated by the same radiologist.

In this series no difference in pulmonary function could be demonstrated between those with and those without spontaneous gastroesophageal reflux.

Tuberous Sclerosis: A Case Report With Simultaneous Respiratory and Renal Failure

Stephen Smith, M.D.; Arnold M. Chonko, M.D. and Gerald R. Kerby, M.D., UKSM-KC

Tuberous sclerosis is a rare autosomal dominant disease that affects primarily the central nervous system and skin. It may, however, involve other organ systems including the heart, kidneys, bones, endocrine system, and lungs. The dominant tissue abnormality is due to angiomyolipomatous tumors. Pulmonary involvement occurs in an estimated 0.1 per cent of cases of tuberous sclerosis. When it is present, the patient often presents later in life with dyspnea or a spontaneous pneumothorax. The pulmonary complications usually dominate the clinical setting and often are the cause of death. When they occur, the incidence of CNS involvement is reduced by 50 per cent.

We report the case of a 39-yr-old female who was first diagnosed as having polycystic renal disease in 1965. Although she had biopsy-proven adenoma sebaceum in 1965, the diagnosis of tuberous sclerosis was not made until 1968 when she required pleurodesis for recurrent left pneumothorax. Since that time, her clinical course has been dominated by recurrent respiratory infections with slowly progressive chronic respiratory failure and cor pulmonale, and chronic renal failure which now requires chronic intermittent peritoneal dialysis.

This woman is unusual for the following reasons: (1) she is not mentally retarded, although she has had seizures and an abnormal brain scan; (2) her clinical course has been dominated by pulmonary and renal insufficiency; (3) she has survived for more than a decade with pulmonary and renal insufficiency; and (4) she is only the tenth person reported with end stage renal failure due to tuberous sclerosis.

RHEUMATOLOGY

Significant Pulmonary Hypertension Associated With Systemic Lupus Erythematosus and Mixed Connective Tissue Disease: A Report of Two Cases

G. D. Schnose, M.D. and J. P. Galichia, M.D., Wichita

The pulmonary manifestations of collagen vascular diseases are numerous and quite variable. In the collagen disorders, systemic lupus erythematosus and mixed connective tissue disease, pulmonary involvement is generally limited to pleuritic and/or parenchymal infiltrates, with disease confined to the pulmonary vasculature being distinctly uncommon and infrequently reported.

We report two cases of pulmonary hypertension in young females, both of whom have known connective tissue disease. The first case is a 27-year-old white female with a seven-year history of systemic lupus erythematosus. This patient's course has been punctuated by numerous flares of arthritis, lupus nephritis, pericarditis, and most recently, progressive dyspnea on exertion. The second patient is a 23-year-old white female with a several-year history of mixed connective tissue disease who presented with acute, severe dyspnea at rest. Right and left heart catheterizations on both patients showed elevated pulmonary artery pressures, both greater than 60/30 mm/mercury, normal pulmonary artery wedge pressures and no evidence of intracardiac shunts. Both patients had pulmonary artery angiograms, which failed to reveal evidence of pulmonary emboli. Echocardiograms demonstrated enlarged right ventricle in each case.

Our two cases offer additional support to the speculation that primary pulmonary hypertension may be a manifestation of certain collagen vascular diseases. Our report includes a review of other possible pathophysiologic mechanisms leading to the development of primary pulmonary hypertension.

Chronic Lymphocytic Leukemia

(Continued from page 28)

Answers

1. d
2. a, b
3. b, c
4. b, c, d

Suggested Readings

1. Catovsky, D.: Hairy-cell leukemia and prolymphocytic leukemia. *Clin. Haematol.* 6:245-268, 1977.
2. Editorial: Radiotherapy of CLL. *Lancet* 1(8107)82, 1979.
3. Knospe, W. H.; Loeb, V., Jr. and Huguley, C. M., Jr.: Bi-weekly chlorambucil treatment of chronic lymphocytic leukemia. *Cancer* 33:555, 1974.
4. Rai, K. R., *et al.*: Clinical staging of CLL. *Blood* 46:219-234, 1975.
5. Rundles, R. W. and Moore, J. O.: Chronic lymphocytic leukemia. *Cancer* (Supplement) 42:941-945, 1978.
6. Sawitsky, A. *et al.*: Comparison of daily versus intermittent chlorambucil and prednisone therapy in the treatment of patients with chronic lymphocytic leukemia. *Blood* 50:1049, 1977.
7. Sweet, D. L.; Golumb, H. M. and Ultmann, J. E.: The clinical features of CLL. *Clin. Haematol.* 6:185-202, 1977.
8. Wiltshaw, E.: Chemotherapy in CLL. *Clin. Haematol.* 6:223-235, 1977.

The President's Message

New Year's Resolutions



It's January and time for resolutions. These are some of mine for 1980:

1. I resolve to spend more time listening to my patients and to listen more attentively, for I know that if I listen closely enough most patients will make the proper diagnosis easy and that many physical symptoms can be relieved by simply allowing the patient to verbalize his or her problems;

2. I resolve to do a thorough examination, for many errors in diagnosis and medical management can be made with hasty conclusions before a thorough examination is performed;

3. I resolve to use laboratory and radiological testing prudently and judiciously so that the proper diagnosis can be made at a minimal expense. However, I will not hesitate to repeat a test or order further tests if clinically indicated. I will also use new techniques when necessary and indicated, and attempt not to use them in lieu of having done a thorough history and physical examination;

4. I will try to be cost conscious, realizing that cost effectiveness can not be equated with human life and that I will not sacrifice quality of care on the altar of cost containment;

5. I will strive always to be professional in my relationship with my patients, my associates, and all those who work with me;

6. I will constantly strive to be gentle in my examinations, tender when necessary in my dealings with patients' emotions, and at all times courteous to my colleagues and my office and hospital personnel;

7. I resolve to continue to practice the best medicine I am capable of practicing and try not to get bogged down with the ever increasing amount of paper work which is present in the practice of medicine today;

8. I will continue to work for the prevention of government intervention in the freedom of medical practice and will do my best to assist my legislators in those issues in the legislature that involve medical practice and human freedom;

9. I resolve to point out to my patients the importance of good health habits and the practicing of preventive medicine wherever possible;

10. I resolve to do my best to make 1980 a good year for all people with whom I come in contact during this year.

Fraternally,

Donald D. Goering, MD

President



Dubious Virtue, Gentle Sin

Now that the season to be jolly has been filed away with the leftover wrappings, we can resume our more characteristic grumpy attitude that goes with the winter blahs. Since the only happy folks these days are those death-wishing people on slats, it is in keeping with this season for our thoughts to turn to some of the less admirable features of human deportment, prominent among which is the sin of opportunism.

We define this as a sin on the word of no less an authority than Dante Alighieri who knew a thing or two about damnation and dismissed opportunists as having "lost the good of intellect." And lest we commit the sin of imprecision, we are referring, of course, to the practice of expedience with consequent sacrifice of principle (which is presumed to give the intellect its necessary exercise). As sins go, opportunism lacks some of the robustness of earthier transgressions, but it makes up what it lacks in quality by its quantity. The boundary between sin and virtue grows dim at times — witness the heart-of-gold prostitute, the benevolent embezzler, the Robin Hoods and Jimmy Valentines. In the case of opportunism, this is particularly true since strict adherence to principle is not always the most rewarding of virtues anyway.

Our current preoccupation with this human failing stems not so much from the winter doldrums as from the recognition — make that resignation — that this will be a highly political year. Politics is often defined as the art of the possible — that is, compromise — and compromise is usually opportunism that has undergone a battle-field conversion. (And any chaplain will advise against counting on such to fill the pews after the shooting is over.) Beside the other electoral activities, the presidential conventions are still six months away, and it seems as if they have been going on for a couple of years. This is largely due to Mr. Carter's anemic showing in sundry polls with its consequent stimulus to intra-party challengers. This has already produced a classical demonstration of opportunism on the part of the senior senator from Massachusetts (by the way, is there a junior senator?) — the feigned reluctance, public exposures aimed at increasing appeal without cutting off retreat if necessary, all orchestrated by an experienced machine adept at detecting all opportunities. Of course, this will not be considered an abandonment of principle since he carries the family conviction that gaining the office is essential to the welfare of the nation.

On the other hand, this — or any — incumbent can invoke the opportunism of statesmanly need to attend to duties rather than descend into the arena when the challenges are inconvenient or difficult. So we can look forward to a continuing policy of expedient behavior from the political world, presidential on down — the prenomination rhetoric fitted with verbal universal joints so it can go any direction, the specious promises of the pre-election campaign, and finally the reality as the survivor explains what he really meant. The degree to which the campaign promise differs from the performance measures the extent of opportunism in the process, and it has always been considerable.

Just because opportunism is the ambient mode in politics, however, we cannot assume that this activity has a monopoly on it. The reliance on expedience is the private property of no single discipline, and our diatribes must acknowledge that medicine, which we

like to think of as the most principled of all, is sustained by expedience to no small degree. We also like to think that it represents a practical accommodation to the realities of medical service rather than an inherent corruption of its practitioners. After all, the practice of medicine is a process of progressive decision making with each decision representing an opportunity for opportunism. The danger lies, of course, in its becoming the operative philosophy in the physician's function, whether intellect is abandoned, as the man said, or never was there. At any rate, we suspect that this transiently comforting but morally debilitating approach is present in the majority of instances in which physicians are found deficient.

However fertile the medical field for such methods, we cling to the belief that they are not characteristic of most practitioners or the character of medical practice. The profession as a whole, however, continues under suspicion because of the utterances of many of its representatives who come on the public scene. Perhaps it is because these individuals attain such status as a result of more or less contact with the political mind, or it may be that the increasing trend toward confronting the medical profession with social decisions leads to statements designed to serve an immediate purpose rather than to support principle. Whatever it is, there is a considerable tendency for these individuals to take the socially expedient approach of putting the profession down by presenting what they think the public wants to hear. For example, one of the syndicated columnists recently called on one of the high organizational spokesmen to respond to a letter of complaint from a patient who had been in a hospital ward and objected to the physical arrangements, the lack of privacy, and general conduct of the service. The response was of the you-are-so-right-we-certainly-should-do-better-we-must-by-all-means-mend-our-ways type. We don't know — maybe it was a pest hole — but the answer made no reference to the economic or practical necessities of providing this level of care nor was any effort made to explain the clinical stringencies that dictate certain conditions or suggest that there was another side. Nowadays, it is not considered good form to stress extenuating circumstance or profess that the presumed deficiency stems from the fact that a given thing is not intended to perform as the critic indicates in the first place. But a reading of these not infrequent confessionals always leaves us wondering where these people have been while these indignities were being visited on a defenseless public. The profession's efforts to fulfill its total obligations have not been so poor that our response to such complaints needs to be a consistent and expedient admission of culpability.

It goes along with an attitude that is distressingly general these days. There are numerous things wrong with the world — whether more in proportion than in past ages, we don't know but rather suspect they are somewhat fewer. But there are still numerous possibilities for humanity or any of its components to perpetrate various evils — especially since new ones are being defined daily. Some of these charges are blatantly opportunistic, and perhaps these do no more than pollute the atmosphere with their querulous recitatives. But a considerable number begin with laudable intent and produce benefits. As the objectionable situation is consequently rectified, the prophets are confronted with putting themselves out of business. The next step then is wholly opportunistic — development of new issues, disclosure of other transgressions, drum beating in new causes — anything but retire to the obscurity that follows a mission accomplished.

Despite the classical assessment and the tone of this presentation, we do admit that opportunism involves a certain form of intellectual exercise (before someone dismisses this effort as editorial opportunism), and our comments suggest an unnecessarily narrow interpretation (as befits such editorial utterances). We don't argue that reliance on a process of opportunistic behavior results in total intellectual atrophy, but, since it is a form of self-service, it inspires similar behavior in others. Thus, when the situation stems from the interaction between two parties, the expedient act may be accomplished by the one who acts first (an opportunism in itself), and the interests of that one receive primary acceptance since the other party is limited to the expedience of reaction.

The current medical scene presents numerous situations that should be considered with these thoughts in mind. For example, unless a patient's access to information is adequate to the decision, only the physician's character and integrity stand against an opportunistic solution. Medical care becomes unjustifiably expensive when it is expedient (especially to a social situation) rather than principled (especially by the medical situation). To the extent that unjustified surgery exists, it is a classical representation of opportunism. So this highly prevalent sin comes down to being a process that does not give appropriate consideration to all the factors and therefore leads to incomplete or inappropriate action.

The use of opportunity is a dubious virtue, and —

(Continued on page 48)

Obituaries

ROBERT GOODWIN RATE, M.D.

Dr. Robert G. Rate, 65, Halstead, died October 25, 1979.

Dr. Rate was graduated from the University of Iowa School of Medicine and served an internship in California prior to entering the Medical Corps of the United States Army. For his innovative work in reconstructive vascular surgery during the Korean War, he was recognized in a Bicentennial publication entitled *200 Years of Military Medicine*. Following retirement from military service, he served as assistant professor of Surgery at the University of Kansas School of Medicine prior to locating in Halstead where he was a member of the surgical staff at the Hertzler Clinic.

Survivors include his wife, two daughters, and two sons. Memorial contributions may be made to the Hertzler Research Foundation Library.

JOSEPH EDWARD RYAN, M.D.

Dr. Joseph E. Ryan, Jr., Emporia, died December 14, 1979.

Dr. Ryan was born in Kansas City, Missouri, in 1912, and was graduated from the University of Kansas School of Medicine in 1936. He had practiced medicine in Emporia since 1947, and had been active in affairs of the Kansas Medical Society.

Survivors include his wife and five sons. Memorial contributions may be made to St. Mary's Hospital Building Fund, Emporia.

RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

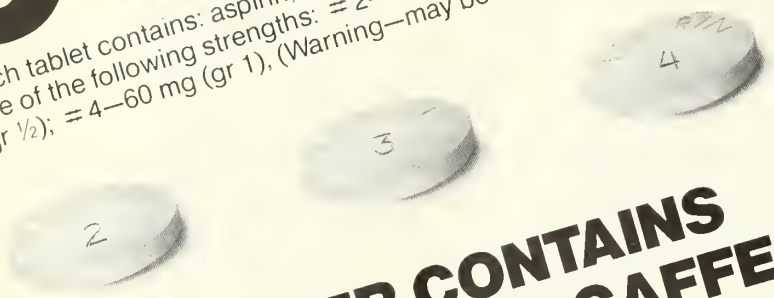
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H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
Roy Neil, M.D., Hays (913) 628-3215
George M. Penn, M.D., Topeka (913) 234-9566
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
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Virginia L. Tucker, M.D., Lawrence(913) 843-3750
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Personalities

Talaat E. Yaghmoor, Pittsburg, was guest speaker at a meeting of the Southeast Kansas Ostomy Association.

David Leitch, chairman of the safety committee of the Garnett Rotary Club, has formulated a proposal to enhance safety for baseball participants in the Garnett park.

William Reals, Wichita, discussed investigation of aviation accidents at a meeting of the Kansas State Nurses' Association, District 10, meeting in Augusta.

Sharfuddin Shah, Halstead, presented a paper, "Cardiomyopathy in Diabetes Mellitus," in St. Louis at the 26th Annual Meeting of the American College of Angiology. He also participated in a panel discussion on "Emerging Concepts in the Management of Diabetes."

Frank Brosius, Wichita, explained the function of the pacemaker for members of Mended Hearts, Inc.

John Travis, Topeka, has been elected president of the American Society of Therapeutic Radiologists.

Wilmer Harms, Halstead, made a slide-illustrated presentation on "Ocular Manifestations of Diabetes Mellitus" to the Harvey County Diabetes Association.

Edgar H. Beahm, Independence, has been listed in the 1979 edition of the *International Who's Who in Community Service*.

Vox Dox

Vox Dox Editor:

I would like to report that the recent Kansas University Medical Center directory that was compiled by the Medical Center, and distributed by the Kansas Medical Society, was incomplete and misleading. Specifically, by design, it included only the activities of the full-time staff.

I would like to inform the members of the Society that a number of us continue to do part-time activities at KU. Specifically, I still have my cerebral palsy clinic there.

W. David Francisco, M.D.
155 So. 18th Street, #105
Kansas City, KS 66102

Vox Dox Editor:

In response to Dr. Francisco's complaint regarding the University of Kansas Medical Center Medical Staff Directory, I wish to explain that this Directory was developed to include only geographic full-time faculty and staff. Voluntary staff, whom I realize play a very important role at the Medical Center, were not to be included in this particular staff directory. I hope, therefore, that he will understand that the omission of his name from this publication was not done intentionally.

Joseph C. Meek, M.D., Director
Health Care Outreach & Continuing Education
University of Kansas School of Medicine
Kansas City, KS 66103

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Dubious Virtue, Gentle Sin

(Continued from page 41)

despite the copybook warning — opportunity knocks — and knocks and knocks. We need to make sure which door it's knocking on and even peruse it through the peep-hole before welcoming it. A matter of principle, you might say. — *D.E.G.*

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Psychiatry and Alcoholism, March 10-12, 1980. Topics will include early identification, etiology, treatment, psychotherapy, physiological factors, Alcoholics Anonymous, research and recovery.

Impaired Physician, Overland Park, May 1, 1980. Cosponsor: Kansas District Branch/APA.

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The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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Division of Hoffmann-La Roche Inc.
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Please see back cover.

Her next attack of cystitis may require

the Bactrim™ 3-system counterattack

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Bactrim has shown high clinical effectiveness in recurrent cystitis as a result of its wide spectrum and distinctive antimicrobial action in the urinary, vaginal and lower intestinal tracts.

The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

Studies have shown that Bactrim acts against *Enterobacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introital colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.

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THE Journal OF THE Kansas Medical Society

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AMA House of Delegates

Summary of Actions taken at the 1979 Interim Meeting, Honolulu, Hawaii

Despite the distance to Hawaii for some delegates, this Interim Meeting was well attended with 272 of the 277 delegates participating in the deliberations. The Kansas Medical Society was officially represented by Donald D. Goering, M.D., President; two delegates and their alternates; and Val Braun, Executive Assistant. Also present from Kansas was William J. Reals, M.D., Wichita, representing the American College of Pathology.

The House considered 74 reports and 98 resolutions on a variety of issues of concern to the practicing physician. Because of space limitations, only those matters which are believed to be of utmost interest to Kansas physicians are reported here. If you would like to have the details of other discussions, please get in touch with one of your delegates listed below.

National Health Insurance

Usually in the forefront of controversial issues before the House, National Health Insurance generated little debate at this meeting. The House is clearly satisfied with the AMA's policy on NHI and swiftly reaffirmed the 1978 principles on National Health Insurance and commended the Board of Trustees.

The House adopted a Board report that called for a continuation of current initiatives of the AMA, so as to ensure that the NHI principles can be forcefully articulated and fully considered in any dialogue that takes place in Congress. Briefly stated, the AMA's policy is to maintain the high quality of medical care within the framework of private health insurance.

Medical Education

The House received a 74-page background report entitled "Future Directions for Medical Education," which proposed sweeping changes in medical education. This comprehensive report addresses issues ranging from admission to medical school and residency training to licensure and evaluation of physicians. It states a need for the restructuring of the third and fourth years of medical school, creation of a "comprehensive residency" for all first-year residents, and establishment of a committee of "public and professional statesmen" to make policy recommendations to the Council on Medical Education.

The delegates filed this report and directed that its suggestions become an agenda of issues for discussion, followed by the development and review of more specific recommendations. This item will undoubtedly be discussed at the 1980 annual meeting (July 20-24).

Delaney Clause Revision

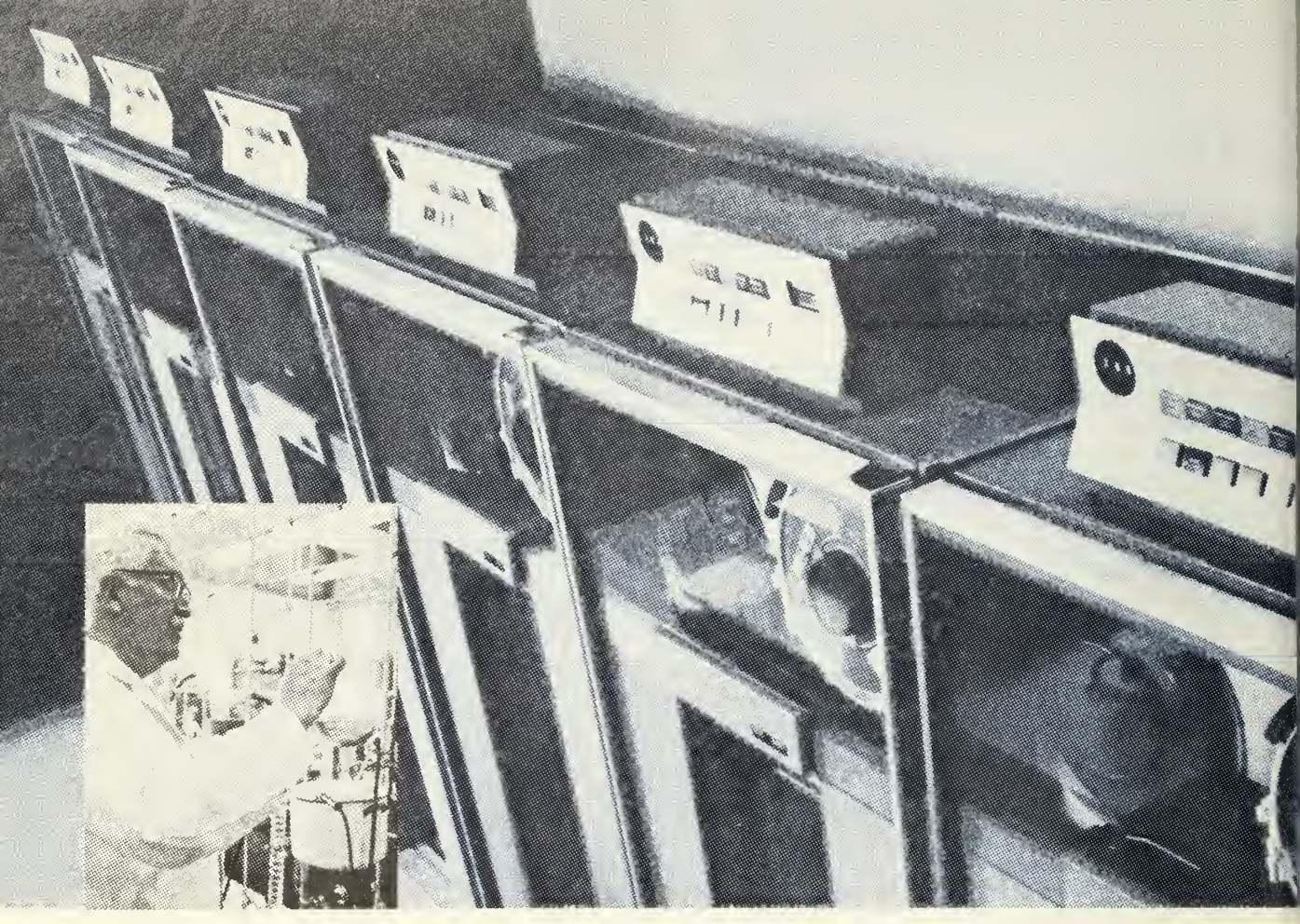
The House adopted an important report from the AMA Council on Scientific Affairs calling for a revision in the Delaney Clause so that (1) Risk/benefit assessments would be made a part of the decision-making process; (2) Alternative risks would be weighed before requiring the banning of a substance expected of being hazardous; and (3) Decision-making on food additives would be based upon the vast scientific evidence available pertaining to the usefulness, function, uniqueness, health needs, and potential risk of the substance.

Membership

Few issues are more frustrating to AMA Delegates and others active in the national association than the continuing problem of recruiting and retaining AMA members. Currently, the percentage of AMA members against the US physician population continues to decline (one hopeful sign in the picture is the modest increase in the numbers of medical students and resident physicians, now totaling over 20,000 in each category). The reference committee reported that in the absence of substantial numbers of new members, association expenses will exceed income in 1981. The high rate of inflation means that the AMA must add several thousand members each year just to maintain the same level of activity. The Council on Long Range Planning and Development is studying various models for restructuring the federation in an effort to resolve the long-term membership problem. According to one report, 42 per cent of the state societies and 63 per cent of the county medical societies have no membership recruitment activities for new members at this time.

If you are not an AMA member at this time, you are seriously encouraged to join now. If a member, you are urged to bring at least one new member into the organization.

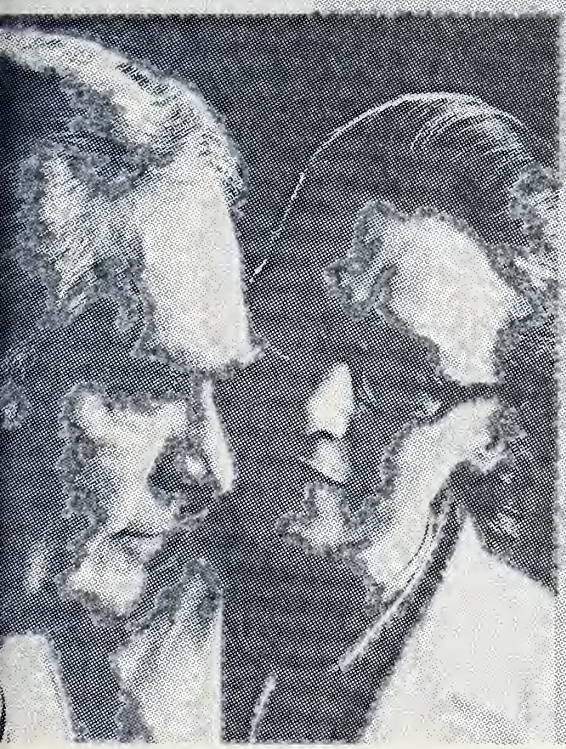
(Continued on page 73)



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- ☐ An unsurpassed safety record
- ☐ Minimal effect on mental acuity, in proper dosage
- ☐ Predictable patient response
- ☐ Is used concomitantly with primary medications, such as anticholinergics and cardiovascular drugs

Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended

Contraindications: Patients with known hypersensitivity to the drug

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults.* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients.* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

Merrell

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Cayey, Puerto Rico 00633

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each tablet contains quinine sulfate 260 mg., aminophylline 195 mg.

specific therapy for painful night leg cramps

Nocturnal recumbency leg muscle cramping is frequently an unwelcome bedfellow for many patients—especially those with arthritis, diabetes or peripheral vascular disease... consider Quinamm... simple, convenient dosage—usually just one tablet at bedtime... can provide restful, welcome sleep without night leg cramps.

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Central Control of Blood Pressure*



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Hyperglycemia	Hyperuricemia
Diabetes mellitus	Gouty arthritis
Bronchial asthma	Sulfonamide hypersensitivity

Like any antihypertensive, use with caution in severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

work/play—normal hemodynamic responses to exercise maintained.

love—low incidence of impotence and/or loss of libido: 2.8% in 1,923 patients studied.[1]

cardiac output—tends to return to control values during long-term therapy.

blood flow—preserved in kidney.

No Single Advantage Determines Drug Choice.

Other factors must include:

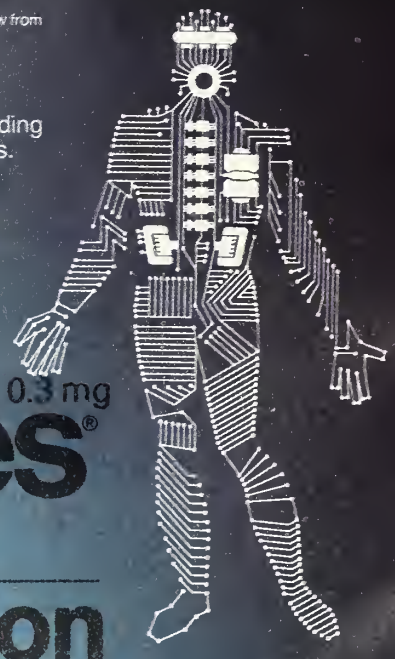
The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

1. Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

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Hypertension



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- No contraindications.
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- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
(clonidine hydrochloride)
Tablets of 0.1, 0.2, 0.3 mg

Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months o longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness an sedation. Constipation, dizziness, headache, and fatigue have been reported Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities i liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlor thalidone and papaverine hydrochloride. Weight gain, transient elevation of bloo glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes nervousness, restlessness, anxiety and mental depression. Also rash, an gioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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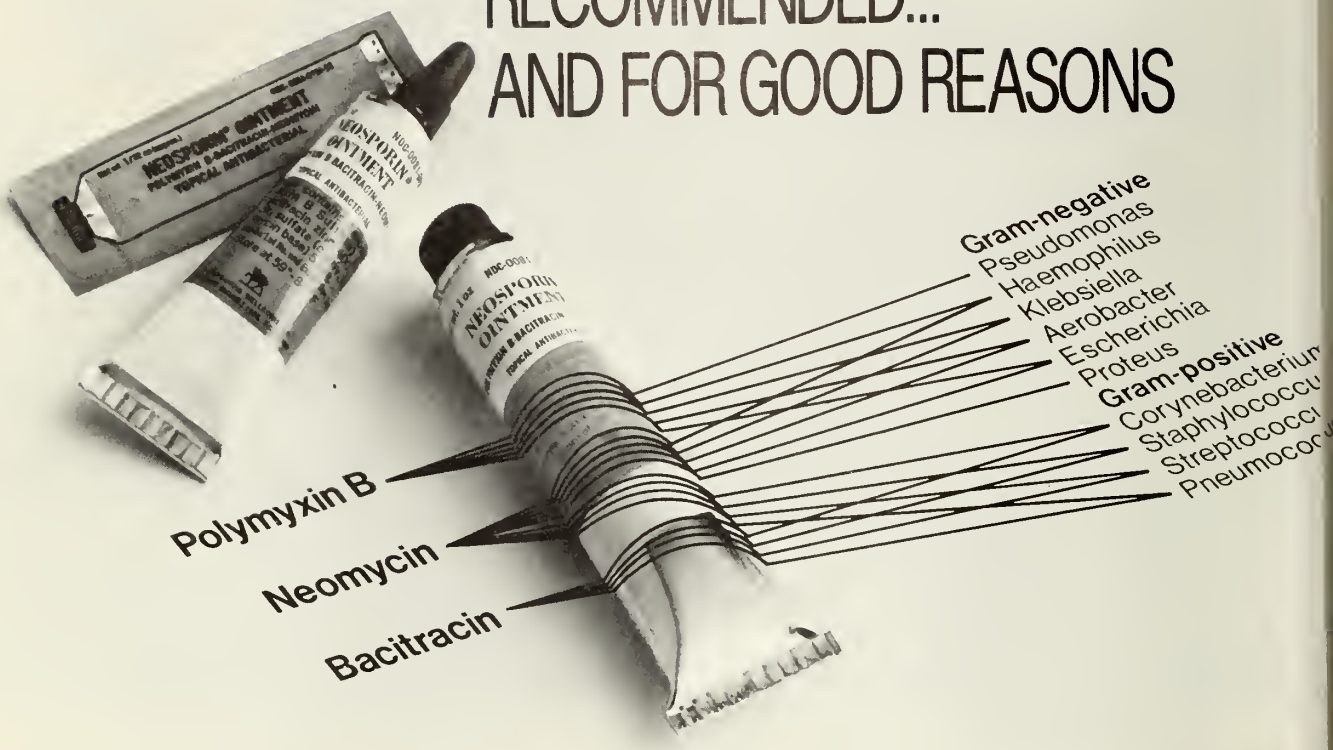
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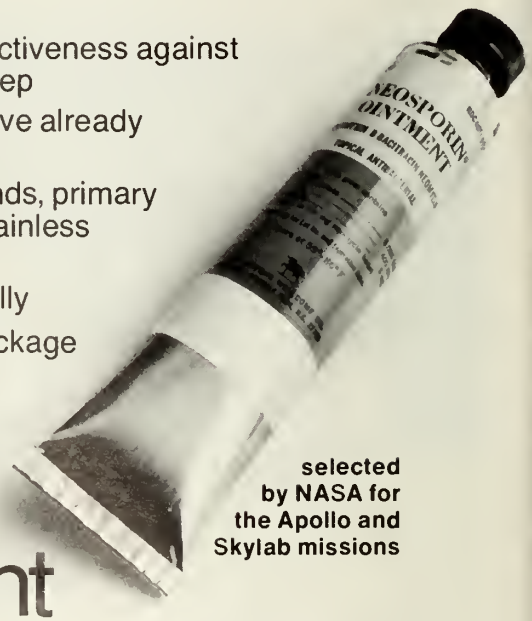
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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter

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KU Executive Vice-Chancellor Dr. David Waxman has indicated to me how much he appreciates the tax deductible contributions of Kansas physicians and spouses, and he is wisely spending the direct contributions in the following areas: student seminars, lectureships, research day activities, volunteer student counseling group, class activities, emergency student loans, visiting professors' lectures, and commencement and graduation expenses.

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AMAERF is a wonderful cause, and with your contributions you are promoting good medicine and helping to ensure the future of medicine. Let's keep Kansas on the map, and if I have touched your heart, please send your contributions either to Peggy Grillot or to me. I am happy to answer any questions, and will welcome all suggestions to improve this very high priority project.

Sincerely yours,
Jean Cavanaugh
Chairman
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Kansas proudly boasts that the AMAERF Chairman for this region is Mrs. Clair J. Cavanaugh, 1320 Cleveland, Great Bend, KS 67530. KMS Auxiliary President Kathy Wedel graciously relinquished her monthly page to allow space for the open letter from Mrs. Cavanaugh, explaining AMAERF and urging support from all Kansas physicians and spouses for the cause.

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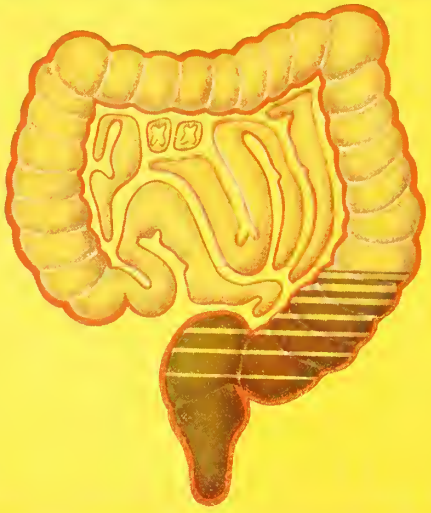
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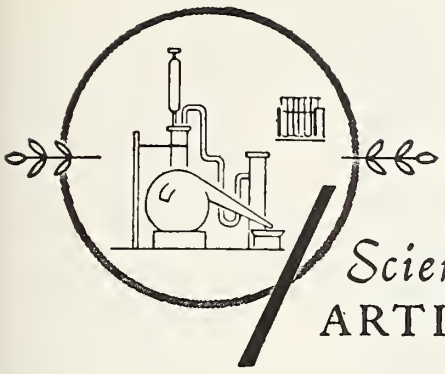
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Cesarean Section

Changing Indications, 1968-1977

RONALD J. BARNHART, M.D., *Shawnee Mission*

Cesarean section rates and indications were compared for two separate years a decade apart — 1968 and 1977 — at Shawnee Mission Medical Center. In 1968, 28 of 729 deliveries were by cesarean section; in 1977, the 209 of 1215 deliveries were sections. Parity, changing indications, average birthweights, forceps incidence, and other factors were compared; and the advent of monitoring and legal influences were considered. Coincident with the increase in cesarean section rate has been a decline in perinatal mortality.

THE RISING cesarean section rate that has occurred nationwide has given rise to increasing concern shared by both professional and lay groups. Various factors have been identified as contributing to this change, but the significance of each of these is still being debated.

Shawnee Mission Medical Center is a 373-bed community hospital. The population that it serves is almost exclusively caucasian and upper middle class. The cesarean section rate at this hospital has paralleled the trend seen nationwide over the past ten years. It is the purpose of this study to compare two years of relatively low and high section rates, 1968 and 1977, to determine what factors have been responsible for this change.

Methods

Statistics were derived from a review of the records of cesarean sections in 1968 and 1977, as well as from labor and delivery room records. Factors considered were: preoperative indications for cesarean section; presence or absence of fetal monitoring; length of labor; parity; and weight of infants. All births for the two years were evaluated with regard to parity, neonatal weight, and the use of forceps.

Results

There has been an inverse relationship between the perinatal mortality rate and the cesarean section rate during the past ten years *(Figure 1)*. The section rates for the two years are indicated in *Table 1*.

In *Figure 2*, the frequencies of the various preoperative indications for cesarean section are compared for the two years. Of the 125 primary cesarean sections performed in 1977, 75 had more than one preoperative diagnosis, as did 14 of 20 primary sections in 1968.

Previous cesarean section was the most common indication for section in both years, noted in 28.6 per cent and 40.0 per cent of sections in 1968 and 1977

Presented at the Kansas City Gynecological Society program, March 15, 1979.

From the Shawnee Mission Medical Center, Shawnee Mission, Kansas.

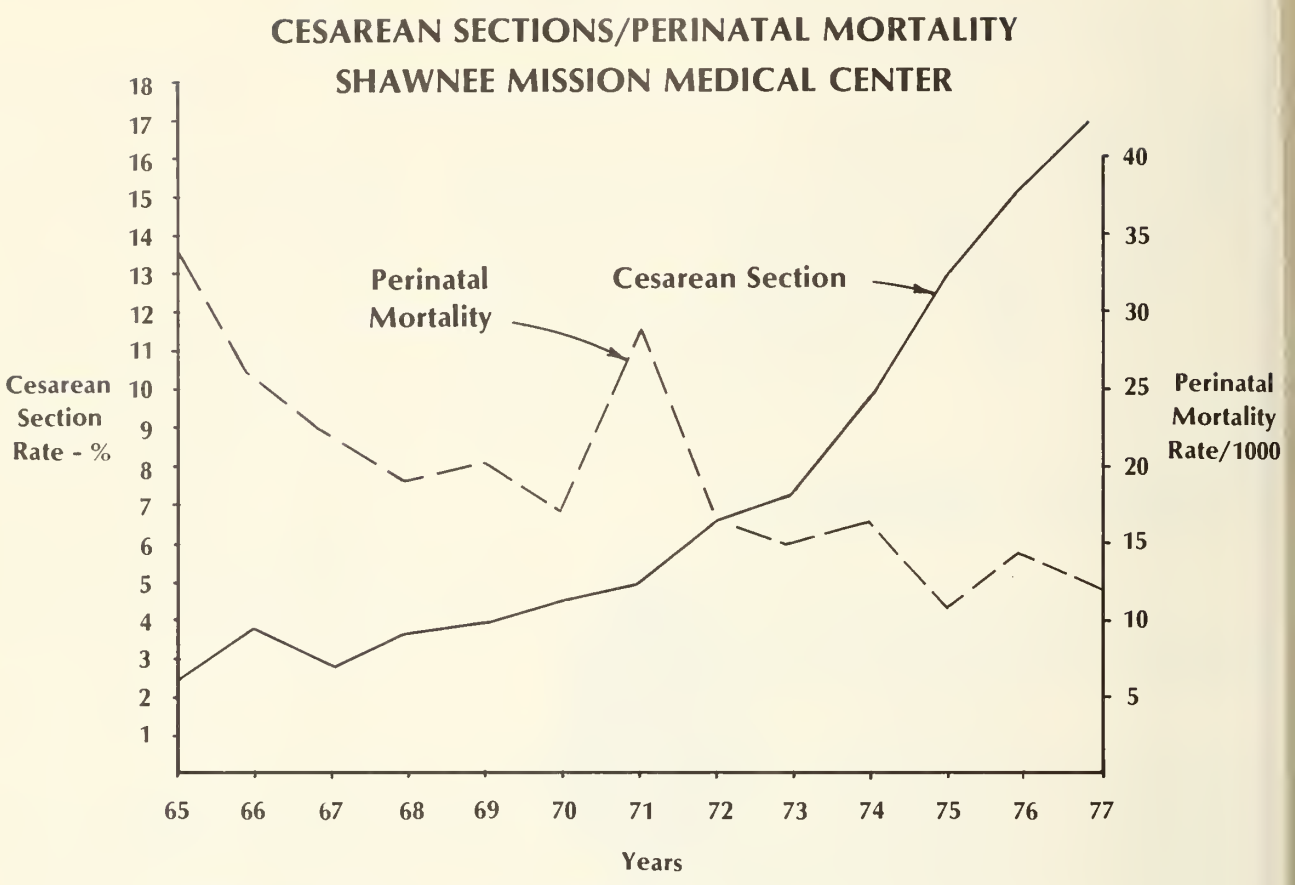


Figure 1. Comparison of cesarean section rate (per hundred births) and perinatal mortality rate (per thousand births).

respectively. The most common indication for primary cesarean section in 1977 was cephalopelvic disproportion (CPD), present in 41 per cent of primary sections. Only one patient out of 729 deliveries in 1968 was sectioned for CPD. Because of this dramatic increase, other related factors were evaluated. The average and term birthweights for the two years as well as the maternal parity are listed in *Table II*. Both the average and term birthweights, as well as the percentage of nulliparous patients delivered, increased significantly.

The frequency of the use of both mid and low forceps was also studied *(Table III)*. Not only was the incidence of the use of mid forceps found to be less than half of what it was nine years ago, but the frequency of outlet forceps application also decreased significantly.

A much higher section rate was found among monitored patients *(Table IV)*. A higher section rate was also found for both CPD and failure to progress among monitored patients, even when those with fetal distress were eliminated *(Table V)*. These two

TABLE I
SHAWNEE MISSION MEDICAL CENTER
CESAREAN SECTIONS

	1968	*1977*
Deliveries	729	1215
Cesarean sections	28 (3.8%)	209 (17.2%)
Primary	20 (71.4%)	125 (60.0%)
Repeat	8 (28.6%)	84 (40.0%)

TABLE II
SHAWNEE MISSION MEDICAL CENTER
BIRTHWEIGHT/PARITY

	1968	*1977*
Average birthweight	3297 gms	3379 gms
Term birthweight (≥38 wks)	3365 gms	3499 gms
Parity		
Nulliparous	270 (37%)	527 (43.4%)
Multiparous	459 (63%)	688 (56.6%)

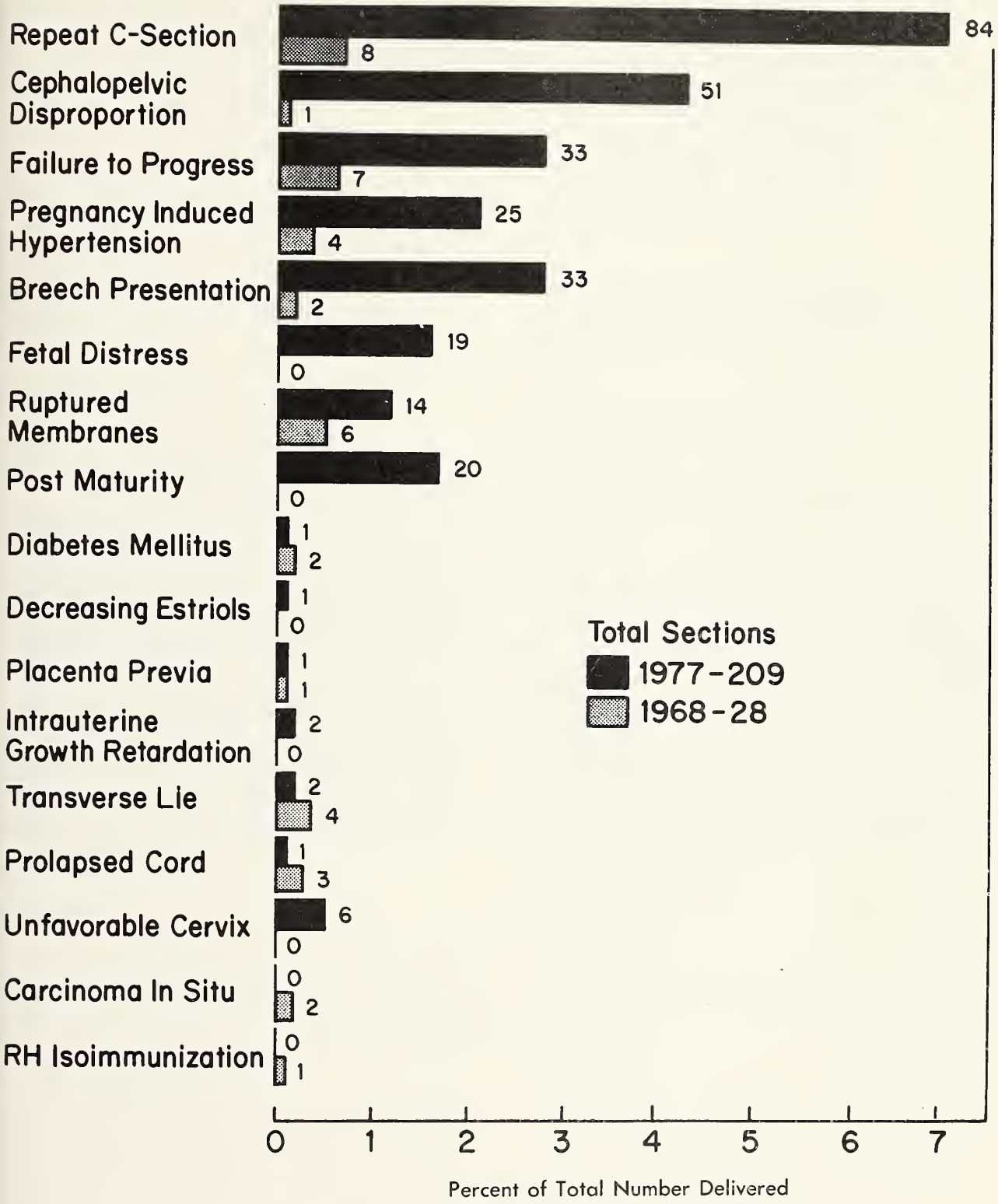


Figure 2. Indications for cesarean sections, calendar 1968, as compared to calendar 1977.

TABLE III
SHAWNEE MISSION MEDICAL CENTER
FORCEPS APPLICATIONS

	Mid Forceps	*Low Forceps*
1968	15/729 (2.06%)	126/729 (17.28%)
1977	11/1215 (0.9 %)	178/1215 (14.65%)

groups were then both studied to determine if length of labor prior to section was affected by monitoring. In both cases labors were longer among monitored patients.

Electronic fetal monitoring was not available at Shawnee Mission Medical Center in 1968. Of the 51 patients sectioned for CPD in 1977, 16 had no trial of labor. Four of the 16 were breech presentations and eight of the remaining 12 had other indications listed as well as CPD.

Thirteen of thirty-one patients with failure to progress were also diagnosed as having CPD, and different physicians appear to have used the two terms interchangeably. Nine of the 31 had a co-diagnosis of prolonged rupture of membranes. Seven patients were sectioned for failure to progress in 1968. All of these had other indications listed.

Fetal distress was the indication for cesarean section in 19 patients (1.56%) in 1977 *(Table IV)*. Sixteen of the 19 were monitored. Ten of the 16 had more than one diagnosis listed as an indication for cesarean section. Fetal scalp sampling was not employed. Fetal distress was found in 9 per cent of the patients sectioned in 1977. There were no patients sectioned for fetal distress in 1968.

TABLE IV
SHAWNEE MISSION MEDICAL CENTER
(1977)
FETAL MONITORING

Monitored	
Direct	94
Indirect	152
Total	246 (20.25%)
Cesarean sections	
Monitored	55 (22.00%)
Not monitored	154 (15.90%)
C-sections for fetal distress	
Monitored	16 (1.30%)
Not monitored	3
Total	19 (1.56%)

Twenty-five patients in 1977 were sectioned with either a primary or secondary diagnosis of toxemia, pre-eclampsia, or hypertension in pregnancy. Eighteen had no labor or attempt at induction prior to section. Of the four patients sectioned for hypertensive disorders in 1968, all had some trial of labor prior to section.

There were 25 breech deliveries in 1968, an incidence of 3.4 per cent. Two (12.5%) were sectioned. In 1977, 33 (50%) of 66 breech presentations were sectioned. The incidence of breech presentation in 1977 was 5.4 per cent.

There were no patients sectioned for postmaturity

TABLE V
SHAWNEE MISSION MEDICAL CENTER
(1977)
CEPHALOPELVIC DISPROPORTION/FAILURE TO PROGRESS

Patients Sectioned for Cephalopelvic Disproportion
(No Fetal Distress)

	Total Patients	*Number Sectioned*	*%*	*Average Length of Labor (Hrs.)*
Monitored	246	19	7.7	10.42
Not monitored	969	25	2.6	6.8

Patients Sectioned for Failure to Progress
(No Fetal Distress)

	Total Patients	*Number Sectioned*	*%*	*Average Length of Labor (Hrs.)*
Monitored	246	21	8.5	13.04
Not monitored	969	8	.8	9.53

TABLE VI
INCIDENCE OF CESAREAN SECTION

	Year												
	65	*66*	*67*	*68*	*69*	*70*	*71*	*72*	*73*	*74*	*75*	*76*	*77*
Evanston Hospital[4]					5.5						10.8		
Women and Infants Hospital Rhode Island[1]	6.9												16.33
University Hospital Ann Arbor[1]													22.9
New York City Hospitals[1]	6.09										14.64		
United States[1]						5.2						11.6	
Mount Sinai Hospital Hartford, Conn.[2]					6.9					10.4			
Booth Memorial Medical Center (Shenker, et al.)	3.3				4.5				6.5				
St. Mary's Hospital, London[6]								8.9	9.7	5.8			
Mayo Clinic[6]	2.7								6.3				
Shawnee Mission Medical Center	2.5	3.8	2.8	3.8	4.0	4.7	5.0	6.2	7.5	9.7	13.3	14.7	17.2

in 1968. All of the 20 patients with a diagnosis of postmaturity in 1977 had other diagnoses as well. Two were breech presentations, and 15 others had the additional diagnoses of CPD, failure to progress, or fetal distress.

Intrauterine growth retardation was the primary indication for cesarean section in two patients in 1977. This diagnosis did not appear in 1968. Other diagnoses seen in 1977 but not mentioned in 1968 were decreasing estriols and cervix unfavorable for induction. Two indications for section seen in 1968 and not occurring in 1977 were carcinoma in situ and Rh isoimmunization.

Discussion

Cesarean section rates have been increasing in both community and university hospitals *(Table VI)*. There has been a corresponding increase in the cesarean section rate at Shawnee Mission Medical Center, particularly during the last five years.

Previous cesarean section, CPD, failure to progress, pregnancy-induced hypertension, breech presentation, fetal distress, and postmaturity are all seen as indications for section much more often now than ten years ago. Forty per cent of sections in 1977 were because of previous cesarean section, as opposed to 28 per cent in 1968.

CPD is now seen as the most common indication for primary cesarean section. An increase in both average birthweight and the percentage of nulliparous patients was found and must be a factor in this change.

There has also been an increasing awareness among physicians of the hazards associated with prolonged or arrested labors, particularly those resulting in difficult forceps deliveries. Mid-forceps were employed less than half as often in 1977 as they were in 1968. It is likely that these patients are now being sectioned for CPD.

Monitored patients were found to be at much higher risk for cesarean section, particularly for CPD. It has been suggested that this occurs because adequacy of labor can be ascertained after less time.[7] However, labors were found to be shorter among those patients sectioned for CPD who were not monitored than among those who were. This suggests that the monitor permitted the physician to allow labor to progress for a longer period of time by knowing the status of the infant at all times. As there is a tendency to monitor those patients considered to be at higher risk — particularly those with prolonged labors such as seen in CPD — it would be expected that these patients would have a higher section rate.

Fetal monitoring has received a great deal of public attention as being the primary cause of increasing section rates nationwide. This was supported by Havercamp in 1976, who found a much higher section rate among monitored patients as compared to a control group, with no difference in perinatal morbidity or mortality.[8] In a study presented by McFee in Kansas City in October 1978, high risk patients were assigned to one of three groups: intermittent auscultation; continuous electronic fetal monitoring; or monitoring with scalp sampling.[7] The monitored

TABLE VII
CESAREAN SECTION
LAC/USC 1970-1976

	1970	*1971*	*1972*	*1973*	*1974*	*1975*	*1976*
Deliveries	9,775	9,425	9,421	10,434	11,585	11,626	13,780
Cesareans	910	822	910	1,068	1,078	1,166	1,468
	(9.3)	(8.7)	(9.6)	(10.2)	(9.3)	(10.0)	(10.7)
Primary	630	573	617	753	763	801	1,019
	(6.4)	(6.1)	(6.5)	(7.2)	(6.7)	(6.9)	(7.4)
Repeat	280	249	293	315	315	365	449
	(2.9)	(2.6)	(3.1)	(3.0)	(2.7)	(3.1)	(3.3)
Monitored patients	1,798	2,337	2,828	3,329	3,746	4,068	4,978
	(18)	(25)	(30)	(32)	(33)	(35)	(36)
C/S in mont. pts.	354	360	465	539	533	579	815
	(20)	(15)	(16)	(16)	(14)	(14)	(16)

groups had three times the incidence of cesarean section with no improvement in perinatal outcome.

This has not been the experience at all hospitals. The cesarean section rate at Los Angeles County Hospital has remained at approximately 10 per cent in spite of the increased use of fetal monitoring *(Table VII)*.[9] At St. Mary's Hospital in London, 20 per cent of labors were monitored in 1972;[5] the section rate for that year was 8.9 per cent. In 1973 and 1974, 85 per cent and 92 per cent of all patients were monitored, respectively. The section rate rose to 9.7 per cent in 1973 but then fell to 5.8 per cent in 1974. It is apparent that the liberal use of fetal monitoring is not necessarily associated with high cesarean section rates.

Twenty-two per cent of monitored patients at Shawnee Mission Medical Center were sectioned in 1977. Only 1.5 per cent of patients were sectioned for fetal distress. Unless the monitor is contributing to the section rate by some mechanism other than fetal distress, it would not appear to be a major factor with regard to the cesarean section rate at this hospital.

As section rates around the country approach 20 per cent, there has been concern that information being obtained by monitoring is being misinterpreted. One solution that has frequently been advocated is the use of fetal scalp samplings to identify those infants truly in distress. It is estimated that 50 per cent of sections for fetal distress could thus be avoided. In this study all but six of the 16 monitored patients sectioned for fetal distress had other diagnoses as well and may have been sectioned regardless of the presence or absence of fetal distress. Even if it is assumed that eight sections would have been avoided with the use of scalp sampling, the section rate would have dropped only 0.7 per cent. It is doubtful that the addition of fetal scalp sampling would have a significant impact on the cesarean section rate at Shawnee Mission Medical Center.

The risks associated with the vaginal delivery of breech presentations are well documented in the literature. Our statistics reflect the increasing use of cesarean section for breech infants. The section rate in this group increased from 8 per cent in 1968 to 50 per cent in 1977. Collea and others have found that term-size infants in a frank breech position with adequate x-ray pelvimetry, no evidence of hyperextension of the head, and not of excessive size may be delivered vaginally without excessive risk.[10] However, legal factors are now such that it is unlikely that the incidence of cesarean section for breech presentation will decrease in the near future.

An increasing number of patients underwent primary cesarean section with no trial of labor in 1977 as compared to 1968. This was noted in 72 per cent of patients with hypertension. Three of these patients delivered infants weighing less than six pounds, and there may have been concern regarding intrauterine growth retardation.

There appeared to be much more concern about postmaturity in 1977, a diagnosis that did not appear among patients sectioned in 1968. This was the primary diagnosis in nine of the 20 patients sectioned for postmaturity. Twelve of these patients were sectioned without an attempt at induction.

The influence of legal considerations in obstetrics is probably the most difficult factor to evaluate, yet it may well be the most important. The cesarean sec-

(Continued on page 78)

Perinatal Deaths

Causes of Perinatal Mortality at Shawnee Mission Medical Center, 1968-1977

EUGENE W. J. PEARCE, M.D., *Shawnee Mission*

PERINATAL HEALTH, as all public health matters, is dependent on both social and biologic factors. It is the biologic factor that we as health care professionals manage in the practice of medicine. Treatment of individual patients occupies our attention so completely that we sometimes fail to see the forest for the trees. Perinatal mortality statistics are a measure, at least in part, of our success in management of obstetrical and pediatric problems related to perinatal health. Consequently, it is appropriate for us to study perinatal deaths and their causes.

Classification and analysis of the causes of perinatal mortality are of value in pointing the direction for efforts to reduce it. The present study was undertaken with the following purposes in mind:

1. To classify (analyze, study) causes of perinatal mortality at Shawnee Mission Medical Center;
2. to discover a technique for continued study of perinatal mortality;
3. to focus the attention of responsible professionals and institutions on manageable causes of perinatal mortality; and
4. to reduce perinatal mortality to an irreducible minimum.

It is not enough to quantify perinatal mortality. It must be studied as a process that can be interrupted, averted, or influenced in order to improve the outcome of pregnancy.

Valdes-Dapena and Arey[1] reported on causes of neonatal mortality by autopsy of 501 newborn infants. Joshi[2] autopsied 100 consecutive cases of perinatal death and listed underlying causes. Naeye[3] reported on 53,518 pregnancies at 12 university-affiliated hospitals in different regions of the United States from a collaborative study done between 1959 and 1966. He personally reviewed the autopsy material from 1435 of the 1993 perinatal deaths in the series. The above studies were based on autopsy material and each author developed his own classification of causes of perinatal mortality.

Manniello and Farrell[4] reported on neonatal mortality in the United States from 1968 to 1974. They used as a system of classification the International Classification of Diseases, Adapted, Eighth Revision.[5]

There were 152 perinatal deaths in 9754 births at Shawnee Mission Medical Center during the ten-year period, 1968-77. All perinatal deaths were reviewed and classified as to cause of death. The main causes were immaturity and placental problems; conspicuously infrequent were isoimmunization and sepsis. The development of a statistical report as a tool for improving perinatal health is discussed.

The ICDA contains a chapter on codes for perinatal mortality. Chapter XV is entitled: "Certain Causes of Perinatal Morbidity and Mortality" (760-779). In addition, Chapter XIV is entitled: "Congenital Anomalies" (740-759). The discharge diagnosis on a newborn infant — alive and well, ill, or deceased — is coded as usual under Chapter XV. In addition, a maternal condition which is a cause of perinatal morbidity or mortality can be coded as such. For instance, pre-eclampsia can be coded both as a maternal condition and as a cause of perinatal morbidity or mortality. There is, then, at hand a working clinical classification for perinatal morbidity and mortality.

In addition, the preface to the ICDA indicates that stillbirth certificate and death certificate diagnoses can be coded using the ICDA and those diagnoses can be used to determine cause of death. The stillbirth certificate in use by the state of Kansas for the period of this study listed cause of death as follows:

(a) Immediate cause:

Presented at the Kansas City Gynecological Society program, March 15, 1979.

From the Shawnee Mission Medical Center, Shawnee Mission, Kansas.

Address reprint requests to Dr. Pearce, 9119 West 74th Street, Shawnee Mission, KS 66204.

TABLE I
AGE OF MOTHER

	Perinatal Deaths #	%	*All Births* #	%
<15	1	0.68	7	0.07
15-19	16	10.96	1047	10.73
20-24	48	32.88	3315	33.98
25-29	53	36.30	3606	36.97
30-34	16	10.96	1361	13.95
35-39	10	6.85	348	3.57
40-44	2	1.37	69	0.71
45-49	0		0	
50>	0		0	
Totals	146		9754	

(b) Due to, or as a consequence of:

(c) Due to, or as a consequence of:

On the certificate is the following statement: "Fetal and/or maternal conditions, if any, giving rise to the immediate cause (a), stating the underlying cause last." The death certificate used the same terminology relating to immediate and underlying causes.

The present study was undertaken on all perinatal deaths at Shawnee Mission Medical Center by reviewing the charts and filling out a code sheet as though the certificate of stillbirth or certificate of death were being filled out by the attending physician. The Medical Records Department at Shawnee Mission Medical Center then coded all diagnoses so stated and the diagnoses were then tabulated and studied.

Materials and Methods

Between 1968 and 1977, the Medical Records Department at Shawnee Mission Medical Center, on request, provided the records of 146 mothers delivered of 152 infants who were either dead at birth or died within the first 168 hours of life. All maternal charts and all paired maternal/infant charts were reviewed personally, and cause of death was assigned and coded as indicated previously in the manner of stillbirth or death certificates. For the most part, the records were excellent and the cause of death obvious.

Considerable information was available from birth certificate data supplied by the Department of Vital Statistics of the State of Kansas. This information was made available to us and was used for comparison. During the study period, the Department of Vital Statistics reported 9754 births at Shawnee Mission Medical Center with 93 stillbirths and 74 neonatal deaths for a perinatal death rate for the period of 17.1/thousand. The material on social status is supplied by the Department of Vital Statistics and the terminology and categories used are theirs.

TABLE II
RACE OF MOTHER

	Perinatal Deaths #	%	*All Births* #	%
White	139	95.20	9614	98.56
Negro	4	2.73	66	0.68
Other nonwhite	1	0.68	73	0.75
Not stated	2	1.37	1	0.01
Totals	146		9754	

Results

The study material was analyzed for age of mother, race of mother, legitimacy and birthweight, and compared to the entire sample for the ten years. This material is covered in *Tables I, II, III,* and *IV.*

The categories for age, race, and legitimacy show no essential differences between perinatal deaths and all births. The expected difference in birthweight between the perinatal death group and all births is quite obvious — 77.0 percent of all perinatal deaths occur in the 2500 gm and under weight group, as compared to 6.8 percent of all births in the 2500 gm and under weight group.

Table V is an analysis of birthweight categories under 2500 gms, by 500-gm increments. Perinatal mortality in Kansas includes all infants weighing 350 gms or over. The Medical Records Department included five infants who weighed less than 350 gms, including three who were stillborn and two who showed some signs of life. Seventy-two (47.36%) of the 152 perinatal deaths weighed less than 1000 gms.

TABLE III
LEGITIMACY

	Perinatal Deaths #	%	*All Births* #	%
Legitimate	133	91.10	9246	94.79
Illegitimate	13	8.90	508	5.21
Not stated	0		0	
Totals	146		9754	

TABLE IV
BIRTH WEIGHT

Grams	Perinatal Deaths #	Perinatal Deaths %	All Births #	All Births %
<2500	117	76.97	662	6.79
2501-4500	33	21.71	8925	91.50
4501>	1	0.66	161	1.65
Not stated	1	0.66	6	0.06
Totals	152		9754	

Of the 87 stillborn infants, 21 were alive at the onset of labor for a percentage of intrapartum death of 24.14 percent of all stillborns, or 13.81 percent of all perinatal deaths.

Of the neonatal deaths, the vast majority — 60 of 65 — occurred during the first 24 hrs of life. Fourteen occurred in less than one hr, 24 in one to six hrs, 11 in 6-12 hrs and 11 in 12-24 hrs. Of the remaining five, three occurred between 24 and 48 hrs, one between 96 and 120 hrs, and in one who died at another hospital, the duration of life was not stated.

Of the 65 neonatal deaths, 59 occurred at Shawnee Mission Medical Center and six at another hospital.

Autopsies were done in 60 of the 152 perinatal deaths. Thirty-seven of the stillborns were autopsied and 50 were not. Twenty-three of the neonatal deaths were autopsied and 42 were not.

Table VI is an analysis of the perinatal deaths associated with congenital anomalies. Quite frequently the anomalies were multiple and only the major one, or ones, were diagnosed and coded. The case of osteogenesis imperfecta (756.6) was associated with multiple congenital anomalies also. It is of some clinical interest that the most common congenital anomaly as a cause of perinatal death is anencephaly (740.0), both as to stillbirth and to neonatal death. As all of these anomalies are quite severe and warrant the classic description "incompatible with life," it would be assumed that this would be an irreducible minimum of deaths due to congenital anomalies. It is of some interest to compare the incidence of perinatal deaths due to congenital anomalies in this series (about 11%) with that of Naeye[3] (9.3%) and Joshi[2] (28%).

Table VII is a classification of all causes of perinatal mortality not associated with congenital anomalies, as coded under Chapter XV, "Certain Causes of Perinatal Morbidity and Mortality," ICDA, Eighth Revision.[5] The diagnoses listed and coded in this table are the underlying causes of death

TABLE V
BIRTH WEIGHT
ALL PERINATAL DEATHS, 1968-77

Weight/Grams	Still-births	Neonatal Deaths
<350	3	2
350-500	8	11
501-1000	25	23
1001-1500	7	8
1501-2000	9	9
2001-2500	7	5
2501-4500	26	7
4501>	1	0
Not stated	1	0
Totals	87	65

as indicated on the certificate of stillbirth or certificate of death. The actual mechanism of death — that is, the proximate cause of death — sometimes is different.

Hypertensive problems in pregnancy (760.3, 762.1, 762.2) are not frequent causes of perinatal death.

Diabetes mellitus (761.1) is also an infrequent cause of death, but several of the cases were Class A diabetes that were underdiagnosed.

Sepsis (763.9) and especially streptococcal sepsis of the newborn (778.9) were notably uncommon.

Attention is called to the very low incidence of death due to respiratory distress syndrome (776.2). However, most of the deaths associated with incompetent cervix (769.0), premature rupture of the membranes (769.1), and immaturity (777.0) were due to respiratory distress syndrome.

The groups under 769 and 777, therefore, have much in common as immaturity is probably the main

TABLE VI
XIV CONGENITAL ANOMALIES (740-759)

	Still Births	Neonatal Deaths	Sub-Total
740.0 Anencephaly	3	3	6
742.0 Hydrocephalus	1		1
746.9 Congenital heart disease		1	1
747.3 Pulmonic stenosis	1		1
753.0 Agenesis of kidneys		1	1
756.6 Osteogenesis imperfecta		1	1
759.8 Multiple anomalies	4	2	6
Totals	9	8	17

TABLE VII

XV CERTAIN CAUSES OF PERINATAL MORBIDITY AND MORTALITY (760-779)

	Still Births	*Neonatal Deaths*	*Sub-Total*	*(Total)*
760.3 Hypertensive cardiovascular disease	1	1	2	(2)
761.1 Diabetes mellitus	4	2	6	
761.5 Fall	1	0	1	(7)
762.1 Pre-eclampsia, severe	4	1	5	
762.2 Eclampsia	1	0	1	(6)
763.9 Amnionitis	1	0	1	
Phlebitis	1	0	1	(2)
766.9 Breech	1	0	1	
768.9 Tumultuous labor	1	0	1	(2)
769.0 Incompetent cervix	2	7	9	
769.1 Premature rupture of membranes	4	9	13	
769.3 Abdominal pregnancy	1	0	1	(23)
770.0 Placenta previa	1	0	1	
770.1 Abruptio placenta	10	3	13	
770.2 Placental infarcts	3	0	3	
770.9 Placental insufficiency	14	1	15	(32)
771.1 Prolapse of cord	0	2	2	
771.9 Velamentous insertion of cord	3	0	3	
Torsion of cord	7	0	7	(12)
775.0 Rh isoimmunization	1	1	2	(2)
776.2 Respiratory distress syndrome	0	4	4	(4)
777.0 Immaturity	0	24	24	(24)
778.1 Postmaturity	1	1	2	
778.9 Streptococcal sepsis	0	1	1	(3)
779.1 Unknown	16	0	16	(16)
Total	87	65		(152)

problem. "Immaturity Otherwise Unclassified (777.0)" indicates that there was no known treatable cause, whereas with the incompetent cervix and premature rupture of the membranes there is at least the possibility of a manageable cause for subsequent pregnancies. Nevertheless, in most of these cases, the mechanism of death was a respiratory one.

Placental problems fall into two main categories. The first is the familiar one of the bleeding complications of pregnancy, including placenta previa (770.0) and abruptio placenta (770.1). Placenta previa obviously is not a major cause of perinatal mortality, and one can assume that it is managed successfully at the present time. On the other hand, abruption still presents a problem. Most of the deaths associated with abruptio were quite sudden and catastrophic and were not amenable to any clinical management — the stillbirths in particular. Analysis of these records indicates that by the time the diagnosis was made, the baby was dead.

The other categories of placental problems — infarcts (770.2) and insufficiency (770.9) — are ones that we recognize more readily today than we did in previous years. It is possible that our heavy emphasis on recognition of intrauterine growth retardation and our program of prenatal testing — including estriol determinations, sonography, and non-stress and contraction stress tests — might be of some value in identifying the fetus that is suffering from placental insufficiency.

Post maturity (778.1) probably should be included under the placental causes and this also would probably be managed with modern technology.

The perinatal mortality due to cord problems is probably at an irreducible minimum. There were three cases of velamentous insertion of the cord

TABLE VIII
CAUSE OF DEATH
STILLBIRTHS OVER 2500 GMS

747.3	Pulmonic stenosis	1
761.1	Diabetes mellitus	3
762.1	Pre-eclampsia	1
762.2	Eclampsia	1
768.9	Tumultuous labor	1
769.3	Abdominal pregnancy	1
770.1	Abruptio placenta	4
770.9	Placental insufficiency	3
771.9	Velamentous insertion of cord	2
	Torsion of cord	5
775.0	Rh isoimmunization	1
778.1	Postmaturity	1
779.9	Unknown	3
Total		27

(771.9) with subsequent vasa previa and rupture of a fetal vessel with exsanguination of the infant prior to delivery. The onset was so abrupt and catastrophic that these cases were not amenable to management.

Torsion of the cord (771.9) as a cause of stillbirth was diagnosed when noted. It is unclear whether this was a cause or a result of stillbirth due to some other cause. It included cord around the neck.

Perinatal death due to Rh isoimmunization (775.0) is of interest in that it is so low. Apparently, the entire array of technology of the last ten years, including Rh immunization, amniocentesis, intrauterine transfusion and improved management of the premature neonate, have all but eliminated isoimmunization as a cause of perinatal mortality.

The 27 stillbirths over 2500 gms demand analysis. *Table VIII* lists the cause of death of all stillbirths over 2500 gms. Deaths associated with diabetes, hypertensive complications of pregnancy, placental insufficiency/post maturity, and isoimmunization appear to be the most likely to be successfully managed with available techniques. Catastrophic or rare conditions including congenital anomalies, abdominal pregnancies and rupture of vasa previa due to velamentous insertion of the cord, are unlikely to yield to any management in the future.

Autopsies were performed on all three of the stillbirths over 2500 gms listed as "unknown."

Table IX lists the causes of death of stillborn infants over 2500 gms dying intrapartum. The three cases of abruption and the two cases of velamentous insertion of the cord were quite sudden and severe

TABLE IX
CAUSES OF DEATH
INTRAPARTUM STILLBIRTHS OVER 2500 GMS

747.3	Pulmonic stenosis	1
768.9	Tumultuous labor	1
770.1	Abruptio placenta	3
770.9	Placental insufficiency	1
771.9	Torsion of cord	1
	Velamentous insertion of cord	2
Total		9

and not amenable to management. The only two cases in this category that might have benefited from fetal monitoring were the one case of placental insufficiency and the one case of torsion of the cord, in which the cord was wrapped around the baby's neck four times.

Discussion

The most common causes of perinatal death in this series are: (1) immaturity, either as a primary cause or secondary to premature rupture of membranes and/or incompetent cervix, and (2) placental problems. A significant number of infants in the immaturity group were in the very low birthweight classification, including those under 500 gms and even a few under 350 gms.

With the present level of skill, dedication and technology in the neonatal special care unit, it would be unwise ever to predict that even the tiniest immature fetus would be impossible to salvage in the future. The obstetrical responsibility under such circumstances is to provide the neonatologist with a warm, dry, oxygenated baby in metabolic balance.

Reduction of perinatal deaths in the placental category is primarily an obstetrical problem as most of these infants were stillbirths. It is probable that at least some of these babies could be saved if present prenatal testing were utilized for babies with suspected intrauterine growth retardation, post-datism, or of hypertensive mothers.

Conditions of low frequency should not automatically be disregarded with the conclusion that they are already at an irreducible minimum. The reduction of just a few perinatal deaths over a decade will, in the future, make a significant difference in the perinatal mortality rate. Specifically, early recognition of Class A diabetes and improved management of pregnancy hypertension, even though both are of low incidence, might significantly reduce perinatal

mortality from both, and thereby, total perinatal mortality.

The present study strongly emphasizes underlying cause of death. As clinicians, our training is to diagnose and treat the underlying cause as a method of dealing with a clinical problem. However, management of the immature neonate is a good example of treating any link in the pathophysiology of the situation to affect its outcome. Treatment of respiratory distress, hypoglycemia, acidosis, cold injury, and the like by the neonatologist will appreciably improve the perinatal outcome in the low birthweight immature group, even if we as obstetricians, knowing the underlying cause, are unable to prevent it.

Another strategy is continual study of the perinatal health. To that end, at the present time the Pediatric Department, Obstetric Department, and Medical Records Department at Shawnee Mission Medical Center have been asked to participate in an effort to develop a statistical analysis of perinatal morbidity and mortality. On January 1, 1979, the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM),[6] was put into use at Shawnee Mission Medical Center. It contains chapters for perinatal morbidity and mortality and for congenital anomalies just as ICDA-8 did, although they are much improved and modernized. The only responsibility of the clinician involved is to be as precise, meticulous, and complete as possible in the diagnosis on the front sheet. Medical Records personnel code all front sheet diagnoses, not only as obstetrical problems, but also as perinatal diagnoses.

Summary and Conclusions

An analysis of causes of perinatal mortality at Shawnee Mission Medical Center over a ten-year period, 1968-1977, using ICDA-8 has been presented. The causes have been analyzed as to their manageability, using available techniques. A proposed statistical analysis based on the use of ICD-9-CM for continuing study of perinatal mortality and morbidity has been presented.

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Vox Dox

Vox Dox Editor:

The December 1979 issue of *The Journal of the Kansas Medical Society* contains an article entitled "Vaginal Voiding" by C. H. Hsu, M.D., Topeka. The article implies that vaginal voiding is pathologically significant in young girls and may be a cause of recurrent urinary tract infection.

Our three-year experience of approximately 300 voiding cystourethrograms performed in the Department of Diagnostic Radiology at the University of Kansas Medical Center on females of all ages fails to support such an implication. Vaginal reflux is found with equal frequency in females symptomatic from urinary tract infection and asymptomatic after treatment. We have not observed it to disappear when infected urine becomes sterile. Furthermore, our experience is identical to that of Shopfner *et al.*, "when voiding cystourethrography is undertaken in the recumbent female, the vagina fills almost invariably during the examination" (Caffey's *Pediatric X-ray Diagnosis,* 7th Edition, Vol. 2).

We must conclude that no pathologic or causative association exists between vaginal voiding and urinary tract infection.

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High Risk Pregnancy

Obstetrics at Shawnee Mission Medical Center

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CLINICAL IMPLICATIONS of the high risk pregnancy concept are dependent upon the ability to identify those at risk and the assumption that a reduction in the associated increased perinatal mortality can be achieved utilizing appropriate and timely obstetrical management.

The high risk pregnancy population is identified by increased perinatal mortality and morbidity over an acceptable value or standard. The frequency of high risk pregnancy is dependent upon the nature of the population served and the definition of high risk pregnancy.

Initially, attempts to define high risk pregnancy involved analysis of conditions relating to pregnancy, labor, and delivery that were associated with increased perinatal mortality. Morbidity has been added to the expanded concept of risk. Ultimately, additional attempts at definition were carried forward to assign a numerical value to conditions or factors associated with perinatal mortality or morbidity so that a prospective predictability could be achieved. Many institutions now employ variations of the Maternal Child Health Index and the Labor Index of Nesbitt *et al.*[1]

According to current literature, about two-thirds of the high risk pregnancy population can be identified in early pregnancy and another one-third either late in pregnancy or at delivery. Further, about 60 per cent of the perinatal mortality arises from the defined high risk pregnancy population while 40 per cent arises from the low risk population.

The data available from the State of Kansas Department of Vital Statistics *Shawnee Mission Perinatal Study* from 1968 through 1977 have been reviewed in an effort to develop information relative to high risk pregnancy at Shawnee Mission Medical Center. There were 9754 births at Shawnee Mission Medical Center between January 1, 1968, and December 31, 1977, weighing 350 gms or more. In this group there were 93 stillbirths and 74 neonatal deaths at 168 hrs or less, for a total perinatal mortality of 17.1/thousand total births.

The data input consists of material submitted with the birth certificate and stillbirth certificate by the attending physician. The death certificates of babies dying in 168 hrs or less of life were matched with their birth certificates. The certificates in use during the period of the study were the United States standard certificates adapted for use in Kansas. The material for analysis is found under:

Risk factors leading to perinatal morbidity were analyzed for 1968-1977 at Shawnee Mission Medical Center. Perinatal mortality rate was 17.1/thousand, with prematurity associated with 72 per cent of all perinatal deaths. Other high risk factors relate to late pregnancy and the intrapartum period. The possible impact on perinatal mortality of prenatal testing and improved neonatal care are discussed.

1. Complications of pregnancy	*Table I*
2. Conditions associated with labor	*Table II*
3. Anesthesia	*Table II*
4. Method of delivery	*Table II*
5. Birthweight	*Table III*
6. Maternal age	*Table III*
7. Plurality	*Table III*
8. Congenital malformations	*Table III*

The frequency of conditions most commonly associated with perinatal mortality are listed in *Table IV* and are compared with normal labor. *Table V* and *Table VI* compile all factors listing actual number, frequency, and perinatal mortality.

Comments

The obstetrical population served at Shawnee Mission Medical Center from 1968-1977 may be described:

Presented at the Kansas City Gynecological Society program, March 15, 1979.

From the Shawnee Mission Medical Center, Shawnee Mission, Kansas.

TABLE I
SHAWNEE MISSION MEDICAL CENTER
PERINATAL STATISTICS
Ten-Year Summary, 1968-1977

Complication of Pregnancy	*Perinatal Mortality Rate*
Pre-eclampsia	12.94
Eclampsia	375.00
Hypertensive disease	34.48
Uterine bleeding not associated with labor	144.14
Bleeding, 1st trimester	100.00
Pyelitis	0
Nephritis	0
Significant anemia	78.43
Significant obesity	43.47
Congenital heart disease	0
Rheumatic heart disease	111.11
Diabetes	79.36
Tuberculosis	0
Syphilis	0
German measles	0
Other viral infections	81.63
Pernicious vomiting	0
Psychiatric or severe psychoneurosis	153.84
Injury or operation	107.14
Malignancy	0
Seizures	0

TABLE II
SHAWNEE MISSION MEDICAL CENTER
PERINATAL STATISTICS
Ten-Year Summary, 1968-1977

Conditions Associated With Labor	*Perinatal Mortality Rate*
Induction of labor	20.06
Normal labor	8.63
Placenta previa	58.82
Premature separation of placenta	379.3
Other intra-partum hemorrhage	115.38
Prolapse of cord	192.31
Breech presentation	72.81
Other malpresentation	37.97
Cephalopelvic disproportion	4.22
Labor 24 hours or more	39.60
Anesthesia	
None	50.59
Local	8.35
Regional	6.69
General	19.66
Method of Delivery	
Spontaneous	15.87
Low forceps	4.97
Mid forceps	22.72
Breech	98.26
Breech extraction	76.19
First C-section	22.19
Repeat C-section	8.44
C-section hysterectomy	0

1. 96 per cent are between 15-34 years of age;
2. 91.5 per cent are delivered of infants with a birthweight of 2500-4500 gms;
3. 6.7 per cent of the infants are premature by weight (<2500 gms);
4. 83.6 per cent of the labors are "normal";
5. 4.22 per cent of the pregnancies were breech;
6. 0.55 per cent of the pregnancies were associated with congenital anomalies;
7. 13 per cent of labors were induced;
8. The total percentage of cesarean sections (primary and repeat) averaged 8.9 per cent. In 1968, 3.5 per cent of deliveries occurred by cesarean section. In 1977, 16.6 per cent of deliveries occurred by cesarean section; and
9. The vast majority of patients are white, educated, and without severe economic deprivation.

The Shawnee Mission Medical Center obstetrical staff consists of family practitioners and specialists. The number of deliveries conducted by each category in this survey has not been determined.

The overall perinatal mortality summary for the

TABLE III
SHAWNEE MISSION MEDICAL CENTER
PERINATAL STATISTICS
Ten-Year Summary, 1968-1977

Birthweight	*Perinatal Mortality Rate*
2500 gms or under	182.78
2501-4500 gms	4.48
4501 or greater	6.21
Maternal Age	
15 or under	142.86
35-39 years old	28.73
40-44 years old	13.16
None over 45	
Plurality	
Twins	73.73
Triplets	0
Congenital Malformations	222.22

TABLE IV

Condition	*Frequency*	*Perinatal Mortality Rate*
Normal labor	83.1	8.63
Induction of labor	13.28	20.06
Breech presentation	4.22	72.81
Pre-eclampsia	3.2	12.94
Cephalopelvic disproportion	2.43	4.22
Twins	2.22	73.73
Birthweight 4501> gms	1.65	6.21
Hypertensive disease	1.49	34.48
Uterine bleeding not associated with labor	1.2	133.33
Labor 24 hours or more	1.04	39.60
First trimester bleeding	1.03	100.00

period 1968-1977 is presented in *Table VII*. A gradual reduction in the perinatal death rate is apparent. The singular factor prematurity by birthweight was associated with 72 per cent of the perinatal deaths. Perinatal mortality associated with prematurity is 183 compared to the average of 17. Of the 667 births of prematurity by weight, 601 were live births, 60 of these dying within seven days for a loss of 10 per cent.

The clinical significance and implications of the high risk pregnancy population has impacted Shawnee Mission Medical Center. Modalities employed to monitor high risk pregnancies have been readily available since 1975. Estriols, L/S ratio determination, sonography, non-stress and stress testing, and fetal heart rate monitors are utilized to various degrees. The obstetrical nursing staff and pediatric nursing staff have undergone added training. Our pediatric colleagues have expanded their expertise and are utilizing the available neonatal intensive care equipment.

The comparison of this data with that from other institutions is tempting. However, the validity of doing so is very questionable. In a general way, the perinatal mortality figures compare favorably to institutions providing similar data. The frequency of risk factors seen at this institution compared to others is different, particularly in relation to diabetes and eclampsia. No data as to the frequency of postdate pregnancy is available from this study, but it is

TABLE V

Condition	*Actual Number*	*Frequency %*	*Perinatal Mortality Rate*
Pre-eclampsia	309	3.2	12.94
Eclampsia	8	0.0008	375.00
Hypertensive disease	145	1.49	34.48
Uterine bleeding not associated with labor	120	1.2	133.33
Bleeding, 1st trimester	100	1.03	100.00
Pyelitis	42	0.43	0
Nephritis	0	0	0
Significant anemia	51	0.52	78.43
Significant obesity	92	0.94	43.47
Congenital heart disease	3	0.00031	0
Rheumatic heart disease	8	0.0008	111.11
Diabetes	63	0.646	79.36
Tuberculosis	2	0.0002	0
Syphilis	2	0.0002	0
German measles	3	0.00031	0
Other viral infections	46	0.00472	81.63
Pernicious vomiting	15	0.0015	0
Psychiatric or severe psychoneurosis	13	0.0013	153.84
Injury or operation	28	0.0028	107.14
Malignancy	7	0.0007	0
Seizures	19	0.0019	0
Induction of labor	1296	13.28	20.06
Placenta previa	51	0.52	58.82
Normal labor	8106	83.1	8.63
Premature separation of placenta	58	0.59	379.3
Other intrapartum hemorrhage	26	0.27	115.38

TABLE VI

Condition	*Actual Number*	*Frequency %*	*Perinatal Mortality Rate*
Prolapse of cord	52	0.53	192.31
Breech presentation	412	4.22	72.81
Other malpresentation	79	0.81	37.97
Cephalopelvic disproportion	237	2.43	4.22
Labor 24 hours or more	101	1.04	39.60
Anesthesia			
None	672	6.89	50.59
Local	3354	34.39	8.35
Regional	2541	26.05	6.69
General	3051	31.28	19.66
Birthweight			
<2500 gms	662	6.77	182.78
2501-4500 gms	8925	91.50	4.48
4501> gms	161	1.65	6.21
Congenital malformations	54	0.55	222.22
Twins	217	2.22	73.73
Method of delivery			
Spontaneous	5860	60.07	15.87
Low forceps	2411	24.72	4.97
Mid forceps	264	2.70	22.72
Breech	173	1.77	98.26
Breech extraction	105	1.08	76.19
First C-section	631	6.47	22.19
Repeat C-section	237	2.43	8.44
C-section hysterectomy	55	0.56	0
Maternal Age			
15 years or less	7	.07	142.86
35-39 years	348	3.57	28.73
40-44 years	76	0.78	13.16

TABLE VII

SHAWNEE MISSION MEDICAL CENTER PERINATAL STATISTICS

Ten-Year Summary, 1968-1977

Total births	9754
Total perinatal deaths	167
Total <7-day hebdomadal	74
Total 20-week+ fetal deaths	93
Perinatal Death Rate	17.12

Year	*# of Births*	*20-Weeks+ Fetal Deaths*	*<7 Days Hebdomadal Deaths*	*Perinatal Deaths*	*Perinatal Death Rate*
1968	737	6	8	14	19.0
1969	827	14	3	17	20.55
1970	942	8	10	18	19.1
1971	884	13	13	26	29.4
1972	924	10	5	15	16.23
1973	1024	11	4	15	14.65
1974	1027	9	8	17	16.55
1975	1040	5	7	12	11.54
1976	1119	8	7	17	15.19
1977	1230	9	7	16	13.01

suspected to be the most common high risk factor seen.

It is apparent that the frequencies of high risk factors associated with perinatal mortality arise late in pregnancy or intrapartum at Shawnee Mission Medical Center *(Table IV)*. For the attending physicians at this institution, the significant questions raised by this data relate to:

1. difficult forceps vs cesarean section;
2. induction of the postdate pregnancy;
3. management of prematurely ruptured membranes; and
4. how best to deliver twins.

Increasingly, these conditions are managed by abdominal delivery and may account for the increased cesarean section rate observed at Shawnee Mission Medical Center.

The incidence of congenital abnormalities of 0.55 per cent in this population is below the average frequency. The perinatal mortality of 222/thousand is impressive. The current emphasis on genetic counseling may introduce additional burdens on the consumers and providers of health care.

The further reduction of perinatal mortality and morbidity may be achieved through intensified effort. Increasing utilization of monitoring modalities and abdominal delivery may impact negatively on the delivery of obstetrical care — a fact that is evidenced by increasing home births.

In summary, the material presented represents an initial effort toward defining the factors associated with poor obstetrical outcome at Shawnee Mission Medical Center. It raises more questions than it answers. The private practitioner of obstetrics and gynecology must be cognizant of the factors associated with increased perinatal mortality and morbidity in the population he serves. Further, more detailed analysis of the specific factors associated with perinatal mortality would be enlightening.

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AMA House of Delegates

(Continued from page 49)

Medicare Reasonable Charges

The House condemned the use of the phrase "reasonable charge" when such use misleads patients and the public. Specifically, the objection was directed at the publication called "Guide to Health Insurance for People with Medicare," published by the National Association of Insurance Commissioners and the Department of HEW. The resolution explained that there was a strong inference that a physician who does not accept assignment and does not accept Medicare's "reasonable charge" as a full payment is, therefore, making "unreasonable charges."

The AMA will investigate this publication and submit a report to the House at the next meeting.

In other actions, the House debated and voted on a wide variety of issues, including:

—Gun Control Legislation
—Health Care for Refugees
—Strip Searching
—Brain Death Legislation
—Treatment of Chronic Mental Patients
—Foreign Medical Graduates

The Kansas Delegation announced to the AMA House the candidacy of Clair C. Conard, M.D., Dodge City, for the AMA Council on Medical Service, and William J. Reals, M.D., Wichita, for the AMA Council on Medical Education. Kansas has thus entered the national arena. In this era of rapid change and constant challenge, adept leadership results not only from a thorough understanding of the issues, but also through a broad conception of constituent attitudes. The Kansas Delegation has full confidence in our candidates' fidelity, industry, and dedication to maintaining the rightful influence of organized medicine in health care policy development. They are considered to be excellent leaders in organized medicine as demonstrated by their experience and performance in professional as well as public service. However, the success of the candidacy of Bill Reals and Clair Conard will be directly proportional to the involvement of each Kansas physician. Much needs to be done between now and July 20, at which time the election will take place. It is hoped that a considerable number of Kansas physicians can attend the Chicago meeting to help campaign for the Kansans. We must be heard nationally!

Please let your delegation members know your views on issues that affect your practice so that we can bring your thoughts to the House. All AMA members are invited to attend the House of Delegates deliberations and express your views at the reference committee meetings.

CLAIR C. CONARD, M.D., *Delegate*
ALEX SCOTT, M.D., *Delegate*
LEW W. PURINTON, M.D., *Alternate*
KERMIT G. WEDEL, M.D., *Alternate*

NST vs CST

The Nonstress Test in High Risk Pregnancies

CRANSTON J. CEDERLIND, M.D., *Shawnee Mission*

OBSTETRIC PRACTICE of the 1970s has found itself centered around the concept of identifying high risk pregnancies and the accurate evaluation of fetal well-being and maturity. The accurate identification of a hostile intrauterine environment and the subsequent proper timing of the delivery of the endangered fetus is paramount in modern obstetrics.

Antepartum fetal heart rate (FHR) monitoring has been increasingly used in the evaluation of the high risk pregnancy. The appearance of late decelerations in the FHR pattern in response to either spontaneous or oxytocin-induced contractions prior to the onset of labor are believed to be related to a decrease in fetal oxygenation secondary to uteroplacental insufficiency.[1] The contraction stress test (CST) also called oxytocin challenge test (OCT) has been widely accepted as a useful clinical method of assessing fetal well-being.

The contraction stress test does, however, have its drawbacks. The test is time consuming, usually requiring from one to three hours of continuous oxytocin infusion with constant attendance by a trained nurse or physician. Conditions such as previous cesarean section, placenta previa, unexplained third trimester bleeding, previous history of premature labor, multiple gestation, premature rupture of membranes, cervical incompetence, and the existence of uterine scars represent certain circumstances where the CST may be contraindicated or may produce further complications.

A viable alternative to the CST has been investigated recently by several authors.[2-9] By observing the characteristics of the spontaneously occurring FHR in response to fetal activity, the concept of the nonstress test (NST) has been developed. The NST is based on the presumption that physiologic acceleration of the FHR in response to fetal movement is indicative of a "good" fetal status. If the fetus accelerates the heart rate in association with vigorous movements, consuming more oxygen and nutrients during a decreased supply period of both to the myocardium, the fetus can be considered to have good reserve. Several authors[2-6, 8] have found that no positive CSTs have been found following reactive fetal heart rate patterns during nonstress tests. The obvious advantages of these findings are many. The NST can be used safely in those situations where a CST would be contraindicated. The NST requires only 15-20 minutes of FHR monitoring without the continuous oxytocin infusion and thus requires less supervision by trained personnel.

The nonstress test (NST) was evaluated as a substitute for the contraction stress test (CST). Eighty-seven NSTs were conducted on 74 patients during a seven-month period (1978-79). There were nine non-reactive NSTs followed by reassuring CSTs. All 74 babies subsequently did well. Therefore, it is concluded that the NST can be used as a screening test for the CST, saving much time and money.

The purpose of this paper is to review the applicability of the use of the NST in the evaluation of high risk pregnancies in a community hospital and review the literature concerning NSTs in combination with subsequent CST testing.

Materials and Methods

Patient selection: During a seven-month period of 1978-79, 87 nonstress tests were carried out on 74 patients at the Shawnee Mission Medical Center. This hospital is a 373-bed community hospital with a total of 1300 deliveries during 1978. *Table I* lists the categories that were used as indications for the use of antepartum FHR monitoring because they are associated with a higher risk for uteroplacental insufficiency.

Technique: Studies were performed by labor and delivery registered nurses and interpreted by a physician. The patient was placed on a bed in the semi-Fowler's position with the head elevated 30 degrees. Blood pressure was measured and recorded at ten-

Presented at the Kansas City Gynecological Society program, March 15, 1979.

From the Shawnee Mission Medical Center, Shawnee Mission, Kansas.

TABLE I
CLINICAL INDICATIONS FOR NONSTRESS TESTING

	No. of Patients
Postmaturity	39
Intrauterine growth retardation	14
Hypertension of pregnancy	12
Essential hypertension	10
Diabetes	8
Decreased fetal movement	4
Third trimester bleeding	3
Elective	3
Elderly primigravida	2
Previous stillbirth	0
Maternal cyanotic heart/respiratory disease	0
Rh sensitivity	0
Sickle cell disease	0

minute intervals to be certain that unrecognized supine hypotension and potential compromised uterine blood flow did not occur. Fetal heart rate and uterine activity patterns were obtained with an ultrasound transducer and a tocodynamometer combined in the Corometric Type III cardiotocograph.

FHR and uterine activity were monitored continuously for 30 minutes. Fetal movement was detected as a transient rise on the uterine contraction channel when confirmed by palpation by the nurse or questioning the patient. Fetal activity was recorded by having the patient press the 50 mm Hg calibration button on the fetal monitor when the fetal movements were felt. To activate the fetus from a possible resting period, efforts to stimulate the fetus by external manipulations were employed every 10-15 minutes if infrequent fetal movements were noted.

Classification of FHR patterns: The tracings were evaluated for the presence or absence of signs of fetal reactivity. Records were analyzed for baseline heart rate, variability, and the heart rate response to spontaneous contractions and fetal movements as outlined by Rochard *et al.*[2] and modified by Everston *et al.*[2] *(Table II).* Heart rate patterns were classified as reactive and nonreactive. A reactive heart rate pattern demonstrated a stable baseline rate usually 120-160 beats/min. The range of baseline variability was six beats/minute or greater, and an acceleration of at least ten beats/minute accompanied fetal movement on at least three movements in the 30-minute observation time. A reactive NST is illustrated in *Figure 1.* Non-reactive patterns were characterized by a stable baseline heart rate 120-160

TABLE II
ANTEPARTUM MONITORING PATTERN CLASSIFICATION

REACTIVE:
- Stable heart rate (120-160 BPM)
- Variability greater than 6 BPM
- Three or more accelerations with fetal movement in 30 minutes

NONREACTIVE:
- Stable heart rate (120-160 BPM)
- Variability less than 6 BPM
- Less than three accelerations with fetal movement in 30 minutes

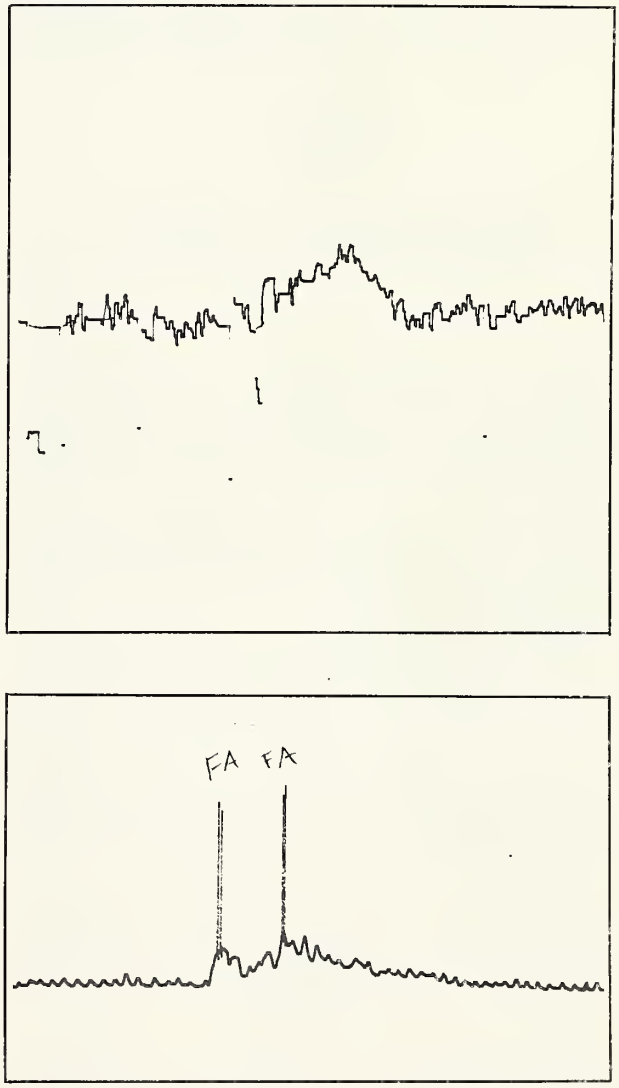


Figure 1. Fetal activity signaled by mother is indicated by spike in lower panel. Irregularity of tracing in upper panel indicates corresponding FHR variability.

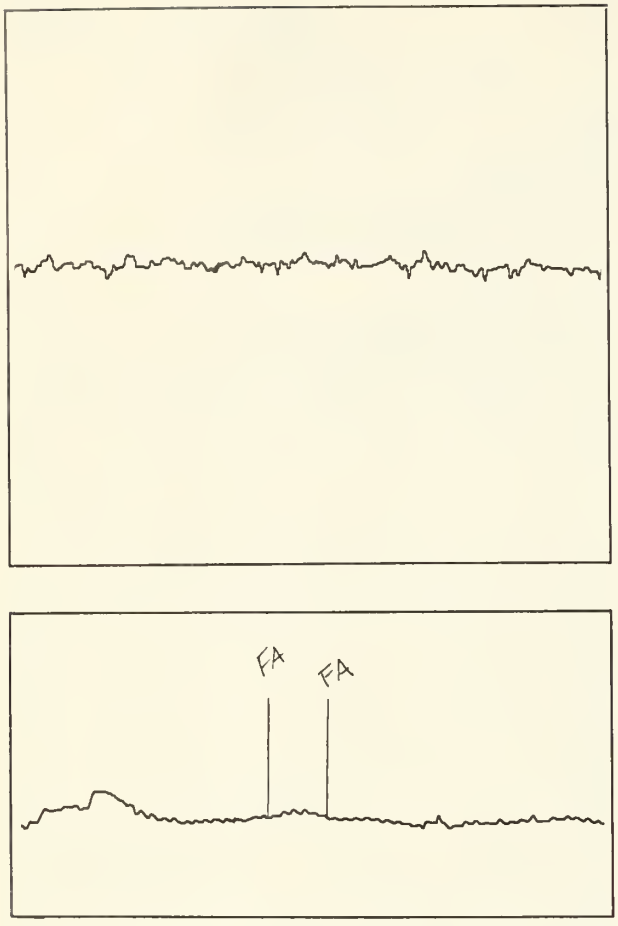


Figure 2. Fetal activity signaled by mother is indicated by spike in lower panel. There is a flat tracing in upper panel indicating loss of FHR variability.

beats/min. However, the variability was consistently less than six beats/min, and there were two or less accelerations with fetal movements. *Figure 2* illustrates a nonreactive NST.

Patients were monitored on a weekly basis. If the patient's FHR tracing revealed a reactive NST, the patient was discharged and a repeat test was performed in one week. If the test was interpreted as nonreactive, a CST was performed. The flow sheet illustrated in *Figure 3* was used as a guideline in the evaluation of the patients.

Classification of outcome: The outcome of each pregnancy was evaluated for: (1) fetal survival or death; (2) presence or absence of fetal distress during labor; (3) one and five minute Apgar scores; and (4) prolonged neonatal hospitalization.

Results: A total of 87 nonstress tests were performed on 74 patients. The earliest gestation was 33 weeks and the latest 43 weeks. The greatest number of tests on one patient was three, least number was one. Of the 87 NSTs performed, nine were read as nonreactive and required subsequent CSTs. All of the subsequent CSTs were read as negative. Of the 74 patients, all delivered within seven days of a monitored test. Seven patients underwent cesarean section prior to labor. Two patients were sectioned for breech presentation. One patient was sectioned for presumed — and subsequently proven — abruptio placenta. One patient was a repeat cesarean section. Two patients were sectioned for progressive hypertension. One patient was sectioned for post dates and an unripe cervix.

TABLE III
RESULTS OF STUDY

Total number of NSTs	87
Total number of patients	74
Reactive NSTs	78
Non-reactive NSTs — negative CSTs	9
Vaginal delivery or c/s with labor	67
Fetal distress during labor	0
Initial and five-minute Apgar below 8	0
Prolonged neonatal hospitalization	0

Sixty-seven patients were allowed to labor. Of these, no patient exhibited any evidence of fetal distress in labor as indicated by either fetal heart rate abnormalities or meconium-stained fluid during labor. Two patients underwent subsequent cesarean sections for cephalopelvic disproportion/failure to progress. One infant had a one-minute Apgar score of five with a subsequent five-minute Apgar score of eight. This infant was one of the breech cesarean sections. Of the other 73 infants, none exhibited an initial one-minute Apgar score below eight. All five-minute Apgar scores were recorded as eight or above. None of the 73 infants required prolonged neonatal hospitalization. The nine NSTs that were read as nonreactive, but had subsequent negative CSTs, exhibited no abnormalities during labor and subsequent neonatal care. *Table III* summarizes these findings.

Comments

This study consisted of 74 patients who were evaluated using a nonstress test to determine the status of their undelivered infants. The study is obviously limited by the small number of patients and the fact that subsequent contraction stress tests were not performed on all patients to substantiate the results obtained from the NSTs. The main purpose of the paper is to demonstrate that a community hospital — which lacks the large patient numbers, adequate personnel and supervised time to conduct a truly relevant study — can nevertheless utilize concepts de-

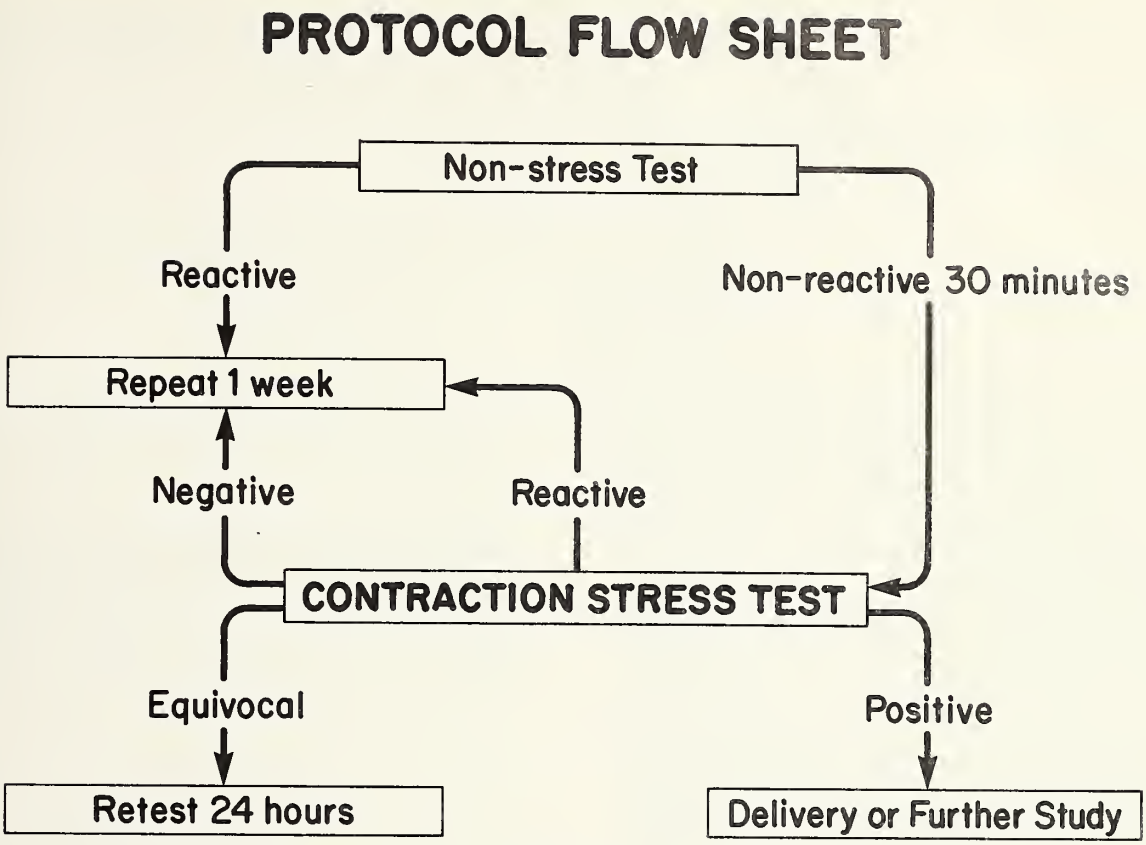


Figure 3

veloped by other authors with those advantages to effectively evaluate high risk pregnancies.

Of the 74 patients tested, none exhibited any signs of fetal distress in labor, all delivered infants with five minute Apgar scores of eight or above, and none could be considered compromised. This relates favorably with the findings of several authors that the nonstress test is an accurate predictor of fetal well-being and that a contraction stress test is not necessary if a reactive pattern is encountered.

Studies by Lee,[5] Trierweiler,[9] and Rochard[8] and their associates correlated contraction stress tests and perinatal outcome with FHR acceleration occurrence. Their findings are summarized in *Table IV*.

These studies indicate that when an adequate number of accelerations with fetal movement were noted during the preoxytocin period of their tests, no patient had a positive contraction stress test during subsequent oxytocin administration. However, when no accelerations of the FHR with fetal movement prior to oxytocin infusion were noted, a positive CST was obtained in from 14-23 per cent of the time. Recently, Evertson, Schifrin, *et al.*,[2] in a study of 2422 NSTs, correlated the number of accelerations in the 20-minute observation time with the results of the subsequent CSTs *(Table V)*. They showed that if the fetus demonstrated two or more accelerations in the first 30 minutes of observation, a negative CST would always follow. Conversely, in 181 patients with no accelerations, 20 (11%) had positive CSTs. In 56 patients with one acceleration, two (3.5%) had positive CSTs. They concluded that: (1) a reactive pattern could be defined as two or more

TABLE V
CST OUTCOME RELATIVE TO NUMBER OF ACCELERATIONS IN THE FIRST 30 MINUTES OF RECORDING[2]

No. of Patients	*Accelerations*	*Positive CST*
181	0	20
56	1	2
36	2	0
25	3	0
4	4	0
302/2422		

TABLE IV
SUMMARIES OF EXPERIENCE WITH NONSTRESS TESTS

Authors	*Total NSTs*				
Lee, *et al.*	324	Reactive NSTs	296	Positive CSTs	0
		Non-reac. NSTs	28	Positive CSTs	6
Trierweiler, *et al.*	255	Reactive NSTs	75	Positive CSTs	0
		Non-reac. NSTs	180	Positive CSTs	25
Rochard, *et al.*	125	Reactive NSTs	76	Fetal death	0
		Non-reac. NSTs	49	Fetal death	16

accelerations in a period of 30 minutes or less; and (2) a reactive NST has a predictive reliability equivalent to a negative CST.

Preliminary experience with the nonstress test suggests that reactive heart rate patterns in the antepartum period indeed reflect fetal well-being. The NST, when employed with the CST as outlined in *Figure 3*, seems to provide useful information as it relates to patient management. Furthermore, the testing procedure proposed is perhaps the simplest form of antepartum heart rate evaluation. Considerable time is saved for the nurse, physician and patient, results are immediately available, expenditures are reduced, patient discomfort is reduced, and patient risk is also reduced due to the many circumstances in which the NST can be employed and the CST may be contraindicated.

Although it is unlikely that any single test involving a biologic system will be associated with absolute predictability, the nonstress test appears to be a highly effective tool for studying the fetus in potentially pathologic situations.

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Cesarean Section

(Continued from page 62)

tion has come to be regarded as the maximum form of therapy in the treatment of most obstetrical problems. It has, therefore, become the method of delivery that exposes the obstetrician to the least risk legally when these frequently encountered obstetrical problems have to be dealt with. In a survey of 50 medical school department chairmen, other professors and selected individual obstetricians throughout the country, almost all mentioned malpractice suits as a factor in increased cesarean section rates.[11]

It is of interest that one of the few reports indicating a fall in the section rate after the institution of fetal monitoring came out of London, England, where concerns of malpractice suits are minimal.

So many changes have occurred in obstetrics in the last ten years that it is unlikely that any one factor can be identified as the sole cause of increasing rates of cesarean section. For the same reason, it should not be expected that a change in approach to any single problem in obstetrics will have a major impact on the section rate. It is possible that the increased use of cesarean section has been a major factor in the dramatic improvement in perinatal morbidity and mortality in recent years. To try to establish the "ideal" cesarean section rate and apply it generally would be to overlook significant variations in patient populations and problems at different institutions around the country.

Risk Birth Registry

A Newborn Screening Model

DENISE BRENNAN COLE, R.N., B.S.N.,* *and*
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A RISK BIRTH registry was started in October 1975 as a pilot project in Wyandotte and Johnson Counties, and was designed to identify and benefit high risk newborns.

Each baby with one or more of four risk criteria noted from birth certificate information was matched according to sex, age, and race with a control baby without risk factors. The risk criteria used were: (1) gestational age of 37 weeks or less; (2) birthweight of 2500 gms or less; (3) respiratory problems during the newborn period; and (4) congenital anomaly. The criteria utilized were based on previous work by Holmes.[1] Follow-up information on growth and development of the babies was obtained from written reports by each child's physician with written permission of the parents. In the event the child showed evidence of developmental problems, information on appropriate community services was shared with the parents through the physician. The results of this program were reported after the first year[1] and on completion of the pilot study.[2]

Though the risk criteria proved satisfactory in discriminating between the children with potential problems and those likely to have few or no problems, a number of practical, logistical problems emerged during the pilot study period. We were not certain that we were reviewing the total number of birth certificates as these were not always immediately available to the county health departments. Some congenital anomalies were not recorded on the birth certificates, the diagnosis apparently either missed or deferred. The number of completed initial contacts with parents was disappointing — less than 20 percent for Wyandotte County babies and less than 40 percent for Johnson County babies. This lack of initial contact continued to be a large problem in spite of concerted efforts to reach parents both by mail and telephone. In addition, the involvement with new parents was too remote to establish the communication with them necessary to explain the program and obtain their signatures on the information release form so that medical information could then be obtained from the child's physician. The developmental ability of each child was not always measured on routine pediatric visits, and the incidence of referrals to stimulation programs could not be assessed.

Critical examination of a risk birth registry pilot study led to the design of a new approach to methodical identification of newborns who are at high risk for developmental problems. This has resulted in a more accurate and complete identification of such infants and better communication with their families, thus improving access to appropriate assistance with health problems.

Because of these deficiencies, it seemed prudent to re-evaluate the entire method of identifying infants who are at high risk for developmental problems, yet attempting to achieve the same goals by more efficient and effective means. The following is a report on a significant outgrowth of the Risk Birth Registry comparing the first year results of a newly established program with those of the former pilot study.[1, 2]

Materials and Methods

Utilizing the recommendations of the Joint Committee on Infant Hearing Screening,[3] the Johnson County Health Department together with the pediatric consultant designed a questionnaire amplifying the basic recommendations to include the original four risk criteria in order to increase the possibility of spotting other developmental problems. In addition,

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Presented at the Kansas City Gynecological Society program, March 15, 1979.

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a question was asked regarding the new parents' interest in parenting classes.

Nine questions were listed and the questionnaire completed by appropriate circling of *YES* or *NO:*

1. Has a close relative of the baby had a hearing loss or any congenital defect SINCE CHILDHOOD?
2. During pregnancy, was the mother exposed to RUBELLA (3-day, German, or "soft" measles)?
3. Was the pregnancy less than eight months?
4. At birth, did the baby weigh LESS THAN 4½ pounds (2000 gms)?
5. Has the baby been SERIOUSLY ill since birth?
6. Does the baby have any defects involving the head, eyes, ears, nose, throat, arms, legs, heart, back, or any other part of the body? Please circle part.
7. Was an exchange transfusion required for jaundice (yellow color)?
8. Do you have reason to be concerned about this child's hearing or other development?
9. Would you like to be contacted about a course in parenthood and parenting?

Use of this newborn screening model was initiated in February 1978, first at Shawnee Mission Medical Center, followed by Olathe Community Hospital and Gardner Hospital. The questionnaires, printed in sets of four copies, are given to new mothers, together with a short letter of explanation and a brochure describing the services of the Johnson County Health Department. The parents are requested but not required to complete the questionnaire. A copy of the completed form is kept in the baby's record, another kept by the parents, and two are returned to the Health Department. The forms are reviewed weekly or biweekly by a Health Department nursing supervisor (DBC) and the pediatric consultant (GEH). Follow-up telephone contact is made by appropriate area nurses of the Health Department, the nursing supervisor and the pediatric consultant, and information added to an on-going central record which is kept of all babies thus registered.

On the follow-up contact by telephone, the mother is asked if there are any problems. If there are none and if she has no particular questions, she is given general information on health department services and parenting classes; the case is then considered closed and so indicated on the central record. If a problem exists, the case remains open until some resolution to the problem has been effected. Every effort is made to strengthen the physician/patient relationship, and in some cases physicians are contacted to offer additional help by means of home visits and developmental evaluations if the physician considers these to be appropriate. Questionnaires on babies residing outside Johnson County are shared with their county health departments after permission to do so has been obtained from the parents.

The nursing staff of one hospital utilizes the questionnaire as a referral form to the Health Department to direct attention to a new mother with particular problems in relating to or accepting her new baby.

TABLE I
ANSWERS FROM NEWBORN SCREENING QUESTIONNAIRE
JOHNSON COUNTY, KANSAS
(2/1/78-1/31/79)

Questionnaires completed	797
No problems or concerns indicated	546
Incidence of affirmative answers to specific questions:	
1. Family history of problems	82
2. Rubella exposure	30
3. Gestation less than 8 mos	18
4. Birth weight < 2000 gms (4½#)	10
5. Baby seriously ill	11
6. Baby born with defect or problem	16
7. Exchange transfusion for jaundice	0
8. Concern for child's development	17
9. Interest in parenting class	131

Results

The total number of children registered during the first year — February 1, 1978, through January 31, 1979 — was 797, with 420 born at Shawnee Mission Medical Center, 348 at Olathe Community Hospital, and 29 at Gardner Hospital.

Table I shows the number of children for whom all answers on the questionnaire were negative (presumably indicating no problems and no requests for parenting classes), and the number of affirmative answers to individual questions.

The first question inquiring about family history problems frequently elicited a positive response which on follow-up usually was found to represent hearing problems diagnosed in adulthood in grandparents or other relatives. Occasionally, parents of the newborn specifically cited a congenital problem in an older child.

Though rubella exposure during pregnancy was noted by 30 mothers, follow-up showed that the

diagnosis was often in question, exposure occurred in the last trimester, the mother had been vaccinated previously, or the mother's physician was satisfied that she had adequate rubella antibody titers. None of the cases had resulted in the birth of a child with suspected rubella syndrome.

Pregnancy of less than eight months was reported only 18 times (2.3%) and low birth weight even less frequently. This is of great concern because these questions focus on the immature and low birth weight groups of babies who are most likely to show developmental problems later. For reasons not entirely clear, we are missing these very important groups.

Possibly also under-reported are the babies known to be seriously ill since birth. However, babies born with obvious defects or other congenital problems appear to be more adequately reported, and these problems are often related to the question regarding parents' concern for the child's development.

No cases of exchange transfusion for jaundice of the newborn were reported, but five babies were noted to have been jaundiced.

The question most frequently answered affirmatively was that regarding interest in parenting class (131, or 16.4% of parents). Occasionally this was misunderstood to relate to available babysitting services. Usually, however, it represented a sincere interest in either attending a class or receiving appropriate material on parenting skills and what to expect of babies at different ages. The *Pierré Pelican* series[4] has proven most helpful in meeting some of these needs. Parents have been given information on existing classes in the community, and in addition, community-wide interest in organizing more parenting classes has been stimulated by the parent response received from this questionnaire.

Of the total 797 babies, at this writing 726 have been contacted by telephone by a member of the Health Department staff; 25 received home visits, five family folders were opened because of continuing concern for the family; 167 (21%) were non-Johnson County residents, and copies of their questionnaires were referred to their respective county health departments, when appropriate, after the initial telephone contact had been made. Problems encountered among the new babies and their parents are noted in *Table II*.

The information obtained by questionnaire was compared with that from the first 100 birth certificates that could be matched by child's name. Only the information referring to the four original risk factors was compared *(Table III)*.

Agreement in the information obtained from the two methods was found on 95 certificates and included reporting from both sources of four congenital abnormalities: congenital hip dislocation, hypospadias, metatarsus adductus, and branchial cleft sinus.

In the remaining five, three were noted from birth certificates to have had respiratory problems (slight or severe asphyxia with resuscitation required) but not noted by parents on questionnaire. Conversely, the other two had abnormalities, one minor (extra digit), one major (Down's syndrome) reported by parents but not recorded on the birth certificates, and thus would have been missed from that source.

TABLE II
NEWBORN SCREENING QUESTIONNAIRE

A. *Problems Encountered Among Babies*	
Myelomeningocoele	1
Down's syndrome	1
Congenital hip dislocation (later talipes equino varus also diagnosed in one)	2
Metatarsus adductus, mild	1
Branchial cleft sinus	1
Deviated nasal septum, mild	1
Hypospadias	2
Extra digits, webbing	3
Fractured clavicle	2
Heart murmur without definite diagnosis	4
Respiratory problems (Rds, pneumothorax, pneumonia)	5
Beta hemolytic streptococcal infection, Group B	1
Hypoglycemia	1
Total	25
B. *Family History Information*	
Parents, one or both, deaf	3
Uncle or aunt deaf since birth	3
Parent with strabismus	1
Sibling with Down's syndrome, other abnormalities, died	1
Sibling with congenital cataract	1
Sibling with seizures	1
Sibling with unilateral hearing loss	1
Family, bonding, marital or parental problems	24
C. *Late Diagnoses (on follow-up contact)*	
Meningitis, eventual hydrocephalus with shunt	1
Cystic fibrosis	1
Pyloric stenosis	1
Strabismus	1

TABLE III
COMPARISON OF INFORMATION OBTAINED BY MATCHING 100 BIRTH CERTIFICATES WITH NEWBORN SCREENING QUESTIONNAIRES

A. *Agreement From Both Sources*	95
Congenital anomalies reported (4):	
1. Congenital hip dislocation	
2. Hypospadias	
3. Metatarsus adductus	
4. Branchial cleft sinus	
B. *Lack of Agreement From Both Sources*	5
Respiratory problems from birth certificate only (3)	
Congenital abnormality from questionnaire only (2):	
1. Down's syndrome	
2. extra digit	

Discussion

In contrast to the original risk registry previously described, this risk program model appears to be more helpful to parents and babies alike, and is more rewarding professionally.

In our comparison of the two methods by matching 100 birth certificates with the questionnaires, we found agreement in the information obtained in 95 cases. In the five cases with lack of agreement, three questionnaires failed to mention the respiratory problems noted on the birth certificates while two others noted congenital problems — one major, one minor — that would have been missed from birth certificate information.

There is good parent cooperation and interest, and the early telephone contact with the parents is gratifying. Health Department personnel answer questions that may be bothering parents regarding feeding, diaper rash, spitting up, cord care, and similar problems. In the event that such problems need more attention, parents are encouraged and urged to see their physician. In addition, the Health Department contacts physicians to offer help without infringing on the doctor/patient relationship.

This program model is a time-saver over the previous approach which involved a vast amount of correspondence and an often frustrating lack of communication. Sharing of information has become easier and more effective as it relates to parents and to other health professionals. Better inter-county communication has resulted from our contacts with the respective Health Departments of non-Johnson County babies.

Though the initial design of the program was to spot the baby who is at high risk for developmental disabilities, it has the additional value of spotting potentially abusive family situations. By utilizing this questionnaire as a referral mechanism, we are able, in a non-threatening manner, to help mothers who need and welcome help but hesitate to admit to such a need.

Some demographic data regarding the mothers are available through these questionnaires, and may be useful in gaining a better understanding of the mothers and families of these newborns.

Responding to the challenge of meeting the two goals of a high risk program, we conclude that we are identifying babies at risk with greater ease than formerly, even though not all parents complete the form. For those who do, we see definite benefits to the babies as well as to their families. Parents and physicians seem less reticent in inquiring about and requesting available services from the Health Department. The interest in parenting classes is noteworthy.

This practical and simplified newborn screening model can be initiated and administered by other interested health departments with comparative ease and efficiency.

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State Meeting
May 2-4
Glenwood Manor
Overland Park, KS



Current COMMENT

New Therapies in the Management of Congestive Heart Failure

FLOYD L. ATKINS, JR., M.D.,* *Kansas City, Kansas*

CONVENTIONAL THERAPY for patients with congestive heart failure is directed at reducing the work load on the heart, improving myocardial function, and decreasing fluid overload. These are achieved primarily with bed rest, digitalis, diuretics, and sodium restriction. These modalities are not always effective; thus there is need for innovative therapies in the management of heart failure. The fundamental determinants regulating cardiac function have been known for many years, but only recently has this knowledge been extended to the medical management of congestive heart failure. Thus, the introduction of vasodilator drugs for use in acute and chronic congestive heart failure is the most important recent advance in the medical management of this entity.

Physiological Alterations in Heart Failure

Congestive heart failure is a descriptive phrase used to define a multi-etiologic condition for which common features are chamber enlargement, low cardiac output, and venous congestion. Classic factors that regulate cardiac action which may be altered in a disease state are the following: contractility of cardiac muscle (inotrophy); heart rate (chronotrophy); *preload* or ventricular filling pressure; and *afterload,* or the degree of impedance to ejection by the heart. The major compensatory response in heart failure is through the Starling mechanism *(Figure 1)* by which increase in ventricular filling pressure produces an augmentation of stroke volume and cardiac output. Patients with heart failure operate on a depressed and flat Starling curve, and the increased filling pressure initiated by renal salt and water conservation does not result in a significant increase in cardiac output, but instead causes pulmonary congestion. Diuretics may relieve this by decreasing vascular volume and ventricular filling pressure, but in doing so may further depress the already compromised cardiac output.

Also in heart failure, because of a decreased cardiac output, there is an effort to preserve blood flow to vital organs. This is associated with enhanced sympathetic tone, high levels of circulating cate-

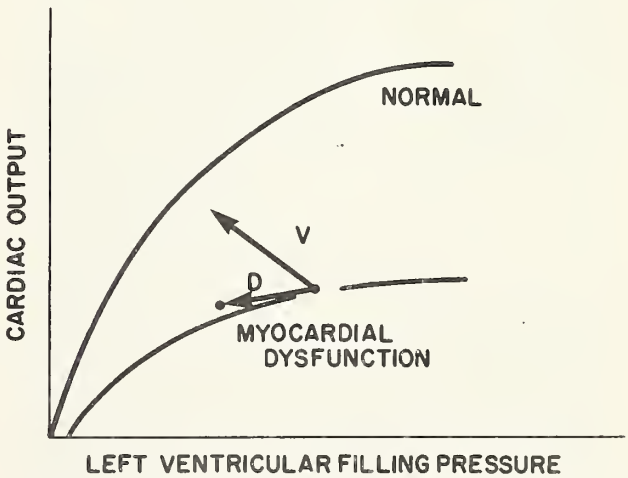


Figure 1. Frank Starling ventricular function curve relating cardiac output to ventricular filling pressure. The depressed curve of myocardial function can be shifted toward normal with vasodilator agents (V). Diuretics (D) may reduce filling pressure causing a decrease or no change in cardiac output.

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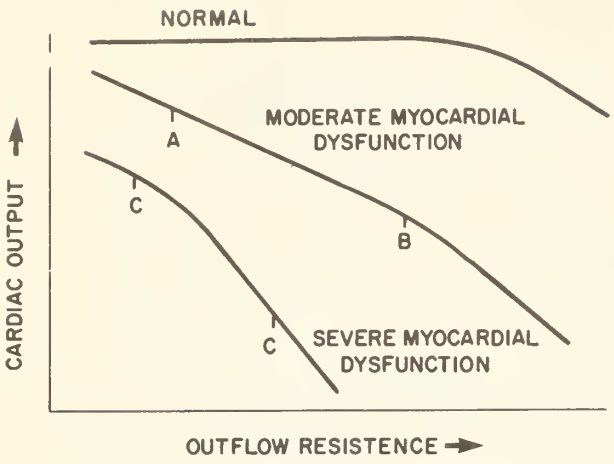


Figure 2. The effect of increasing the systemic resistance in the normal and diseased hearts. Cardiac output is maintained in the normal heart even at high levels of outflow resistance. At increasing degrees of myocardial dysfunction, increases in outflow resistance cause significant reductions in cardiac output.

cholamines, and increased activity of the renin-angiotensin system. These factors are responsible for the increased systemic vascular resistance which increases impedance to left ventricular ejection leading to a depression of cardiac output. In the normal heart, increased outflow resistance is of little consequence, but in patients with myocardial dysfunction, the institution of vasodilator therapy to lower impedance can produce dramatic improvements in cardiac performance *(Figure 2)*.

Strategy in Use of Vasodilator Drugs

Vasodilators have been classified as venodilators and arterial dilators. Arterial dilators either increase the compliance of the large arteries or relax the arteriolar resistance vessels. These reduce aortic impedance (afterload), allow the dysfunctioning ventricle to empty more completely, and enhance cardiac output. Venodilators increase the capacitance of the systemic venous bed and lead to a redistribution of intravascular blood volume from the central to the peripheral reservoirs. Venodilators reduce left ventricular filling pressure (preload) and relieve pulmonary congestion. Nitroprusside is still a widely used and effective vasodilator agent but since it must be given intravenously with continuous hemodynamic monitoring, its use has been limited to the intensive care unit. Now a variety of oral, sublingual, and cutaneous agents are available for use in ambulatory patients. These drugs provide a spectrum of action that results in the increase of lowered cardiac output by reducing peripheral vascular resistance or a decline of increased ventricular filling pressure by decreasing venous tone. Thus, each drug can modify cardiac function differently, depending on the alterations of preload venous impedance, which are dependent upon the relative effects on systemic resistance and venous capacitance vessels characteristic of each agent.

The ratio of change in forearm vascular resistance to change in venous tone reflects the degree to which an agent affects arterial resistance when compared to its effect on ventricular filling pressure *(Figure 3)*. Until recently, the most widely used vasodilators have been the long-acting nitrates and nitroglycerin. These agents primarily affect the venous capacitance vessels with only a slight decrease in peripheral vascular resistance. The associated fall in ventricular filling pressure relieves the symptoms of pulmonary congestion. Impressive declines of increased left ventricular filling pressures result for four to six hours following the sublingual (5-10 mg) or oral (20-40 mg) administration of isosorbide dinitrate. In addition, cutaneous nitroglycerin ointment (1-2 inches) provides sustained action similar to the oral long-acting preparations.

The need for an oral long-acting vasodilator principally to increase lowered cardiac output in chronic heart failure has led to the use of oral hydralazine (25-100 mg, 3-4 times/day). The potent direct systemic vascular relaxing effect is confined to the arterial bed, resulting in an increased cardiac output. In patients with symptoms of low output and pulmonary congestion, hydralazine is best used in combi-

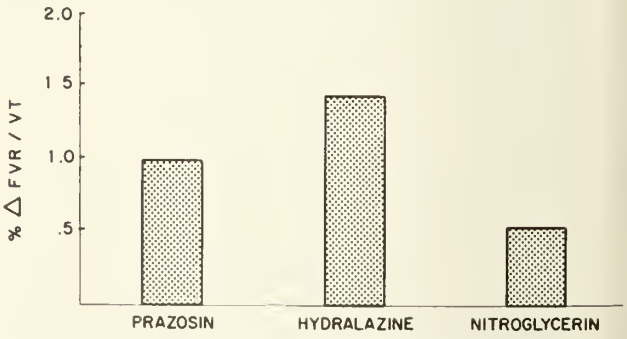


Figure 3. Effects of vasodilator agents on the ratio of the percentage change in forearm vascular resistance (FVR) to percentage change in forearm venous tone (VT). A ratio of one indicates a balanced effect with equal arteriolar and venous relaxation as seen with prazosin. Hydralazine produces greater arteriolar relaxation and nitroglycerin results in predominately venous dilatation.

nation with long-acting nitrates. The most recent vasodilator agent introduced is prazosin, which has an equal effect on the venous and arteriolar systems. Its acute effects on the circulatory system result in a reduction of both afterload and preload with an increase in cardiac output and a decrease in ventricular filling pressure. Ambulatory therapy with prazosin (2-7 mg, 4 times/day) has been effective in the treatment of refractory heart failure.

Complications of Vasodilator Therapy

Although some reduction in arterial pressure is expected during vasodilator therapy, marked reduction could lead to complications. In patients with only borderline elevations of left ventricular filling pressure, the administration of nitrates may decrease the filling pressure below the level necessary to maintain an optimal cardiac output *(Figure 4)*. However, in most patients with chronic heart failure, there is only a modest or no reduction in arterial pressure. Hydralazine therapy may be associated with undesirable side effects. Some of the reported minor side effects include headache, nausea, and skin rash. The lupus-like syndrome is the most serious side effect and occurs in 10-20 per cent of patients taking a daily dosage of 400 mg or more. Prazosin, while initially met with enthusiasm, has been found by some investigators not to have persistent beneficial hemodynamic effects. This is not true of long-term hydralazine or nitrate therapy and emphasizes the need for continued clinical evaluation of individual patients on prazosin.

Summary

While digitalis and diuretics have been the mainstay of therapy for congestive heart failure, recent innovations have been introduced. Vasodilatory therapy is based on pharmacological alterations in the determinants of cardiac function. Agents such as the nitrates predominantly lower ventricular filling pressure by decreasing venous return and relieve symptoms of pulmonary congestion. Conversely, hydralazine decreases systemic resistance, resulting in more efficient emptying of the left ventricle and an increase in cardiac output. Prazosin has a balanced effect on venous tone and systemic resistance and, therefore, decreases ventricular filling pressure and increases cardiac output. The use of these agents in combination with digitalis and diuretics has added greatly to our ability to treat chronic heart failure in an ambulatory setting. While we have been able to document symptomatic improvement with these agents, the effects of vasodilators on long-term mortality remain to be determined.

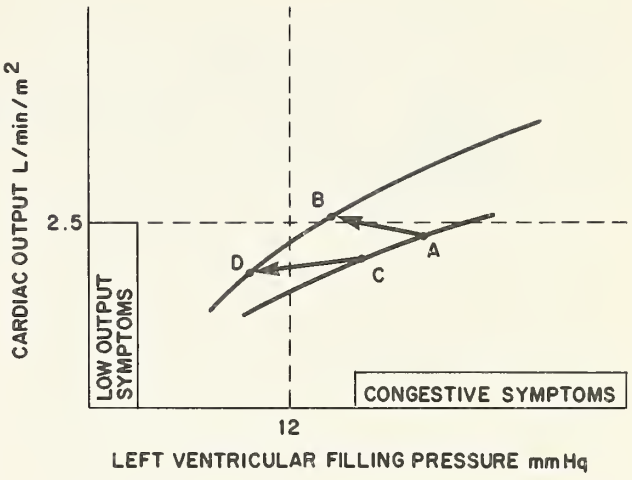
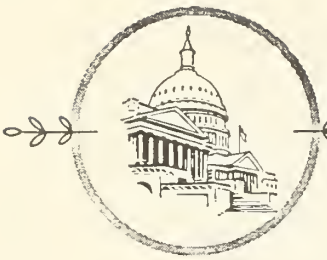


Figure 4. The effect of nitrates on cardiac output and left ventricular filling pressure in two patients with low output and congestive symptoms. Patient A with the higher elevation of filling pressure had both an increase in cardiac output and relief of congestive symptoms (B). In patient C, the filling pressure was decreased below the normal, resulting in a fall in cardiac output (D).

Self-Assessment Questions

1. Patients with congestive heart failure generally have which of the following:
 a. Enlarged heart
 b. Decreased cardiac output
 c. Increased left ventricular filling pressure
 d. All of the above
2. The major compensatory mechanism utilized by the failing heart involves Starling's law. (T or F)
3. Nitrate preparations have their *major* effect on which of the following:
 a. Venous tone
 b. Arterial resistance
 c. Myocardial contractility
 d. None of the above
4. Prazosin has a balanced effect in heart failure patients and decreases ventricular filling pressure as well as it decreases peripheral arteriolar resistance. (T or F)
5. Hydralazine may have which of the following side effects:
 a. nausea and vomiting
 b. Skin rash
 c. Lupus syndrome in doses over 400 mg/d
 d. All of the above

(Answers on page 94)



Socio-ECONOMICS

Effective Records Management

The four-drawer filing cabinet is as outdated in modern medical practice as the application of a leech to a patient. The new open-shelf, color-coded, lateral filing method is both convenient and economical.

Why Convert?

- In most offices, staff salaries account for 15 per cent of gross practice revenues. In other words, staff time is expensive. With a four-drawer filing cabinet, a great deal of this expensive staff time is spent opening and closing drawers in the filing and retrieval process. With lateral filing systems the medical assistant need not open and close the drawer every time a file is pulled or refiled; the open view and color guides him/her to the correct spot.
- It has been estimated that it costs approximately 17 cents to file a chart and $65 to find a misfiled chart. Color coding drastically reduces misfiles.
- Files that are not color coded take considerably longer to file and to retrieve.
- Rental space accounts for 5 per cent of gross practice revenues in most areas. Four-drawer filing cabinets take up 50 per cent more space than open-shelf lateral files. This space can usually be utilized more effectively for other purposes.

How to Convert

- *Open-shelf filing.* Open-shelf lateral filing can be easily compared to a bookshelf. Files are lined up on an open shelf to a height of approximately eight feet from the floor. The name or number appears on an easy-to-scan end tab. The open shelves can accommodate two to three feet more files in height than a four-drawer cabinet, and the shelves are better space savers than drawers.
- *Section dividers.* A section divider is a thick cardboard or plastic file divider with an end tab or top tab that marks the beginning of a section. In an alphabetical file, three to five section dividers will be utilized per letter in the alphabet. Section dividers serve to single out those areas in which the largest number of files are situated. For example, in an office with 20 or 30 patients with the last name of Johnson, a special section divider might be implemented for that last name. In this way, the file search for "Johnson" is limited by the divider. Naturally, section dividers should be customized to the alphabetic breakdown of patient names in each practice. Section dividers can be purchased for about 20 cents each.
- *Color code.* This inexpensive technique limits the file search and consequently saves staff time. In most systems, eight basic colors are used. For example, a half inch red band is affixed to the tab of all "A" files; another color (like orange) to all the "Bs" and so on until each of the eight colors has been used; the colors are then repeated in the same order. Most offices code charts by the first two letters of the patient's last name; thus, in a code system

Medical records management has progressed a long way from the four-drawer filing cabinet invented in 1893. Physicians no longer have to bear the many aggravations of an antiquated record-keeping system. With the implementation of an open shelf, lateral, color-coded filing system, the medical practice will reap rewards of efficiency in time and money for many years into the future. Anyone can file a record; the trick is to find it.

where blue represents "J" and yellow represents "O," a Johnson chart will be marked with blue and yellow.

Color coding can also be effectively utilized in numerical systems to limit file search and eliminate misfiles.

• *Out-guides*. An out-guide is a larger colored file, usually thicker cardboard or plastic colored yellow or blue for easy recognition. When a medical record is pulled from the shelf, an out-guide can be inserted in its place. When refiling the chart, the medical assistant can zero in on the general area of the file and then look for the out-guide. This, too, reduces refiling time.

Out-guides with plastic pockets may be utilized so that letters, reports, or miscellaneous paper can be filed in the out-guide on a daily basis. In this way, the general refiling of routine reports will not be disrupted if the file is not on the shelf.

Out-guides can also be purchased with a preprinted log on which to note the date the file was pulled and who has it. This establishes greater control over the file and allows it to be located quickly in an emergency.

• *Alphabetical sorting*. Medical assistants should be trained to sort files in alphabetical or numerical order before refiling. Once the files are in proper order, the assistant need only scan for the out-guide and replace the files in correct order starting from one end and moving down the line.

• *Inactive charts*. Generally if a patient has not been in within five years, the chart should be pulled from active files and put in a less accessible space or a low-cost storage area.

The initial culling process should be done during a weekend and all charts should be "weeded out" at that time. To facilitate the continuing process, a color-coded dot can be affixed to each file to designate a patient visit in a particular year. For instance, if a red color dot denotes the year 1980, a red dot will be affixed to the chart of any patient who visits the practice during 1980. If the patient returns in 1981, a yellow dot may be placed on the chart. In this way, when the time comes to cull the charts, the colored dots will quickly indicate which patients have not been seen during the previous five years. All staff should know the color dot code by year.

Inactive charts should be filed alphabetically in a storage area. Since they will not often be needed, they can be filed in a lower-cost storage area away from the office, or in non-prime space.

THE UNIVERSITY OF KANSAS COLLEGE OF HEALTH SCIENCES AND HOSPITAL

DIVISION OF HEALTH CARE OUTREACH AND CONTINUING EDUCATION

SYMPOSIA:

PEDIATRICS

March 10, 11 and 12, 1980

Guest Faculty:

RAMON CANENT, M.D., University of Missouri—Kansas City School of Medicine and The Children's Mercy Hospital.
LAWRENCE M. GARTNER, M.D., Albert Einstein College of Medicine, Bronx, New York.
RONALD M. LAUER, M.D., University of Iowa, Iowa City.
GEORGE H. McCRACKEN, JR., M.D., The University of Texas Health Science Center at Dallas, Southwestern Medical School.
GERALD L. MANDELL, M.D., University of Virginia School of Medicine, Charlottesville.
LLOYD C. OLSON, M.D., The Children's Mercy Hospital, Kansas City, Missouri.
ROBERT J. ROBERTS, M.D., Ph.D., The University of Iowa College of Medicine, Iowa City.
ABRAHAM RUDOLPH, M.D., University of California Medical Center, San Francisco.

Subjects to be discussed will include: THE INFANT IN CONGESTIVE HEART FAILURE; EARLY RECOGNITION OF LIFE-THREATENING CARDIOVASCULAR ABNORMALITIES; THE SIGNIFICANCE OF BLOOD PRESSURE MEASUREMENT IN CHILDHOOD; THE SIGNIFICANCE OF CHOLESTEROL LEVELS IN CHILDHOOD; HYPOXIA — RECOGNITION AND MANAGEMENT; RETROLENTAL FIBROPLASIA; KERNICTERUS AND ITS POSSIBLE PREVENTION; RENAL HANDLING OF DRUGS BY THE NEWBORN KIDNEY; WHAT'S NEW IN INFANT FEEDING — IF ANYTHING; CONTROL OF INFECTIONS ACQUIRED IN THE FIRST MONTH OF LIFE; USE OF ANTIBIOTICS IN NEONATES; PHAGOCYTE FUNCTION IN HEALTH AND DISEASE; CHLAMYDIAL INFECTIONS.

Accreditation:
American Medical Association: 17 hours of Category 1.
American Academy of Family Physicians: 17 hours of Prescribed C.E. Credit.

Registration Fee: $135.00.

PROSPECTS IN REHABILITATION MEDICINE FOR THE 1980's

March 26, 27 and 28, 1980

Guest Faculty:

JEAN COLE, Ph.D., Texas Institute for Rehabilitation and Research, Houston.
GABRIEL FAIMON, M.P.A., Rehabilitation Services of Kansas, Topeka.
ROBERT C. HARDER, Th.D., Kansas State Department of Social and Rehabilitation Services and University of Kansas School of Social Welfare.
ISAAC K. JOHNSON, B.S., M.A., Region VII Rehabilitation Services Administration, Department of Health, Education and Welfare.
HERBERT KENT, M.D., Department of Rehabilitation Medicine, Veterans Administration Hospital, Long Beach, California.
PAUL LEVY, President, The Whole Person, Kansas City, Missouri.
HOWARD MOSES, B.G.S., Division of Vocational Rehabilitation, Department of Social and Rehabilitation Services, Topeka, Kansas.
CHARLES R. PETERSON, M.D., Howard A. Rusk Rehabilitation Center and University of Missouri School of Medicine, Columbia.
LOIS SCHWAB, Ed.D., University of Nebraska, Lincoln.

Subjects to be discussed will include: REHABILITATION ENGINEERING — A KEY TO THE FUTURE OF REHABILITATION; THE SEVERELY NEUROLOGICALLY DISABLED — NEW APPROACHES; TRANSPORTATION FOR THE DISABLED — OPTIONS AVAILABLE; INDEPENDENT LIVING FOR SEVERELY DISABLED — THE NEBRASKA EXPERIENCE; INDEPENDENT LIVING FOR SEVERELY DISABLED — THE KANSAS PROGRAM; PHYSICAL AGENTS; EXERCISE AND GAIT TRAINING; OCCUPATIONAL THERAPY; SPEECH PATHOLOGY AND AUDIOLOGY; PAIN CONTROL METHODS; PROSTHETICS AND ORTHOTICS; THE INDEPENDENT LIVING MOVEMENT: IMPLICATIONS FOR REHABILITATION MEDICINE.

Accreditation
American Medical Association: 14½ hours of Category 1.
American Academy of Family Physicians: 14½ hours of Prescribed C.E. Credit.

Registration Fee: $95.00.

For program announcements and information, write: DIVISION OF CONTINUING EDUCATION, The University of Kansas College of Health Sciences and Hospital, Kansas City, Kansas 66103

The President's Message

Official KMS Policy



So you'd like to have an influence on the policies of the Kansas Medical Society! Well, now is the time and here is how you can do it.

As you know, the policies of the Medical Society are formulated by the House of Delegates. The House of Delegates meets annually, and in 1980 this will be on May 3 and 4, at the Glenwood Manor in Overland Park, Kansas. The House of Delegates makes its policies by voting on resolutions. These resolutions can be presented by any member of the Medical Society, any of the component societies, or any of the specialty societies represented in the Kansas Medical Society.

If you have a resolution that you would like to present to the House of Delegates, please prepare your resolution at this time (if you need help in how to formulate a resolution, please contact the Medical Society executive office). Once your resolution is in proper form, please forward it to the Kansas Medical Society office before March 15, so that it can get into the April issue of the *Kansas Medical Society Journal* and will then be available for discussion at the State Meeting.

Resolutions have already been submitted regarding chiropractors, physician supervision of physician's assistants, the voluntary effort, prospective rate review, and several other topics. However, more resolutions are needed, and we solicit your participation in this policy-making process.

Each year we have some late resolutions which are not submitted in time for the April issue of *The Journal*. This year we would like to keep them to a minimum if at all possible. In this way, all the Medical Society members have a chance to know what topics are going to be discussed and formulate their opinions about these topics.

Remember, *you* can do it. Your Medical Society can do it. Your specialty society can do it. And we at the Medical Society office urge you to do it. So let's do it now before March 15 if at all possible.

Then at the State Meeting each of you will have the opportunity to discuss your resolution as well as other resolutions before the reference committees. This is true even if you are not a delegate to the House of Delegates. In this way each member of the Medical Society can have an impact on official Kansas Medical Society policy.

Fraternally,

Donald D. Gerring, MD

President



Present Tension

Stress represents mankind's oldest physiologic insult. As a direct manifestation of the civilizing process, this conversion of the adaptive stimulus to a state of damaging pressure is imposed by the very successes of the process. The 20th Century has brought maturity and recognition to stress as a way of life, a dubious tribute to the benefits of leaving the simpler ways. Inevitably, however, its establishment as a bona fide entity has spawned a reciprocal concept known, so far, as under-stress. As you might surmise, the ill effects of this newly-admitted condition are the result of the organism's being subjected to less pressure than it needs to reach optimum productivity. This will undoubtedly come as a source of comfort in some quarters — Uncle Dudley, for example, who can now be referred to as understressed rather than just a slob as has been the case heretofore.

Before this new concept diverts our attention from the traditional interpretation of stress completely, we should note that, after these many years in which it has been integrated into virtually every pathologic entity, stress has sponsored several free-standing forms which should be reviewed as primary conditions rather than secondary influences on other diseases.

Credit Cardiopathy: The real nature of this disorder has become apparent only recently since it was first interpreted as an abnormal attachment to plastic. The basic connection was not appreciated since the clinical manifestations usually did not develop for one to two months after the contact with the plastic, at which time a cumulative effect characterized by hostile behavior, verbal rantings comparable to speaking in tongues, a strong denial pattern, but primarily a plaintive, accusatory attitude of the patient toward his/her family becomes apparent. If uncontrolled, the condition appears to increase in severity at the rate of 18 per cent a year.

The name is derived from the German physician, Karl Credé, who first enunciated the principle known as "puschen kommt bei shoven" to express the fundamental law that if one puts enough pressure on one end, something is bound to come out the other. Unfortunately, the obstetricians appropriated the term, and their misuse of it through the years has clouded the fact that it represents perhaps the most fundamental and succinct statement of stress.

Though clinical features are varied, patients are generally all charged up. Therapy extends over many months, and it is not uncommon for new attacks to develop before earlier ones are relieved, so the patient may actually show different stages of a number of episodes at the same time. This can lead to the malignant form which requires radical excision of the offending plastic, but generally it is possible to keep the condition under control if the patient keeps up his monthly treatment sessions (known in therapy circles as the "E-Z" plan for stress reduction).

Reader's Digestive Dysfunction: This is a stress phenomenon stemming from an abnormal reaction to print. Attacks are precipitated by exposure to publications of an inspirational character, such as those describing individuals who become international business tycoons canning chili in their basements or 90-year-old grandmothers who obtain three degrees and an exterminator's license in night school in addition to doing windows. The attacks are charac-

terized by nausea, epigastric burning, and alternating anger and depression. It should be pointed out that these symptoms are of a general nature, being associated with a variety of disorders. However, careful analysis will disclose that the patient may represent a paradoxical form of stress reaction — that is, actually under-stress, or they wouldn't be reading the articles in the first place.

Again, a malignant form is recognized, this being produced by reading the alumni bulletin which reports that the new library at the Alma Mater will be named after the principal benefactor (naturally) who happens to have been the class jerk and whose attendance at the dedication ceremonies will represent his first visit to any library.

Therapy has so far been only palliative and limited to removal of the patient from the irritants. One instance of cure was reported in a patient who sold an article to a magazine describing how he didn't amount to anything and enjoyed it. It was later discovered, however, that the patient was a hermit, and he was disqualified on the basis of professionalism. In the meantime, the only accepted criterion of improvement in the condition is a feeling on the part of the patient that he wants to lick the world — beginning with chili canners, 90-year-old grandmothers, and the class jerk.

Kannicht-Neinsagen Syndrome: In their classical paper in the Allgemeine Zeitschrift für All Sorte Dingen (Germans are very big in stress research — it's the language), Kannicht and Neinsagen demonstrated that the condition previously known as *egotropia exalta* was, in fact, a clear-cut example of stress. These patients have a biochemical defect which, when they are confronted with a request for a contribution of time or money, renders them incapable of refusing. This really involves two levels of stress, the first phase created by the request which the patient does not recognize as such since it inevitably leads to acquiescence. Since he has been assured that he is being approached because he is (1) an outstanding leader in the community; (2) an essential element in the civic and charitable life of the community; or (3) possessed of superlative judgment and unique capabilities, he experiences a form of anesthesia, so that the second phase of stress, the necessity of fulfilling the obligation, becomes apparent too late for him to extricate himself. The diversion of his resources may arouse some objections by his family, thus aggravating the stress since he has to justify his actions (plus an unspoken anger that said family doesn't seem to recognize all those qualities in him). Between the first and second phase, there is a brief transition in which a spontaneous immunization (indicated by the sudden blurting of an agonized "No!") may occur and establish protection against the second phase. This is by no means a life-long immunity, and the usual result from exposure is a sensitization that increases the patient's susceptibility to subsequent attacks.

This first stage is known on the street as getting hooked. Once in this situation, the victim often seeks relief by involving others in such activities and thus graduates to the role of "pusher." There is no effective treatment, and the patient often becomes paranoid since he finds he is either being sought by those seeking his services or avoided by those who know he will put the bite on them for something (another street term). Despite the lack of consistently effective therapy, the authors were able to report one spontaneous cure in a patient who served on a committee to abolish fundraising campaigns which ran out of money.

Amnesia natalis: This is one of the most common and more debilitating forms of stress in terms of duration and impact on behavior. It is insidious in onset, often becoming apparent by age 20 and reaching a fixed stage by age 30, and may be heralded by annual episodes of agitated depression and denial. A strong ambivalence is noted in the early stages as the patient seeks to enjoy the attentions associated with his birthday but avoid the aging implications. Thus, the patient has a normal time-space orientation except for this inability to acknowledge that he shows any evidence of change. Because of his conviction that his own age is remaining static while that of others is advancing, he develops an elaborate pattern of behavior to support it. Instead of going to the barber shop for a haircut, he patronizes a hair salon for a styling. This is a primary part of the condition since any tendency toward decrease or graying of hair precipitates a painful crisis known as trichopanic. Since the patient is unduly sensitive to constrictions about the throat, he affects open collars, the depth of the opening being determined by the severity of his affliction, the amount of hair on his chest, and the length of his neck chain.

Hyperactivity, interpreted by the patient as an expression of youthfulness, is common. One form is precipitated by music with the response being directly related to the decibels. He pursues numerous forms of physical exercise, especially those calling

(Continued on page 94)

RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 625-7049
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
Roy Neil, M.D., Hays (913) 628-3215
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*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences–National Research Council and/or other information, FDA has classified the following indications as "probably" effective:

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS. Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. WARNINGS. In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS. Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS. Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include: xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations, mydriasis; cycloplegia, increased ocular tension; loss of taste, headache, nervousness; drowsiness; weakness; dizziness; insomnia, nausea; vomiting; impotence, suppression of lactation, constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis, urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION. Dosage must be adjusted to individual patient's needs.

Usual Dosage. Bentyl 10 mg. capsule and syrup: *Adults*: 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children*: 1 capsule or teaspoonful syrup three or four times daily. *Infants*: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults*: 1 tablet three or four times daily. Bentyl Injection: *Adults*: 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE. The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

Product Information as of October, 1978

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New Therapies

(Continued from page 85)

Answers

1. *d*
2. *True*
3. *a*
4. *True*
5. *d*

Suggested Readings

1. Symposium on Vasodilator and Inotropic Therapy of Heart Failure (Mason, D. T., ed.). *Am. J. Med.* 65:101, 1978.
2. Packer, M. and Meller, J.: Oral vasodilator therapy for chronic heart failure: A plea for caution. *Am. J. Cardiol.* 42:686-689, 1978.
3. Cohn, J. N. and Franciosa, J. A.: Vasodilator therapy of cardiac failure. *N. Engl. J. Med.* 297:27-31, 254-258, 1977.
4. Arnold, S. B. *et al.:* Attenuation of prazosin effect on cardiac output in chronic heart failure. *Ann. Intern. Med.* 91:345-349, 1979.

Present Tension

(Continued from page 90)

for distinctive costume and conducted in public view. His behavior pattern obviously separates him from his contemporaries and prompts him to seek companionship in progressively younger circles, particularly feminine. If he survives this obviously hazardous course, he goes into the chronic state since there is no active therapy. He usually succumbs still retaining his convictions and sometimes because of them. In rare instances, a spontaneous remission may occur in later years when, as the patient views himself in the mirror some morning, the amnesia is inexplicably replaced by a sense of humor.

The condition has never been reported in the female.

This review is part of our effort to provide every opportunity for continuing medical education, one of the newer stress conditions to be reported. However, in recognition of under-stress, no self-assessment test is included. — *D.E.G.*

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Warnings: Serious, occasionally fatal, anaphylactoid reactions have been reported. Some patients with penicillin hypersensitivity have had severe reactions to a cephalosporin; inquire about penicillin, cephalosporin, or other allergies before treatment. If an allergic reaction occurs, discontinue the drug and treat with the usual agents (e.g., epinephrine or other pressor amines, antihistamines, or corticosteroids).

Precautions: Use with caution in individuals with histories of significant allergies and/or asthma. Do not rely on oral administration in patients with severe illness, nausea, vomiting, gastric dilatation, cardiospasm, or intestinal hypermotility. Occasional patients will not absorb therapeutic amounts given orally. In streptococcal infections, treat until the organism is eliminated (minimum of ten days). With prolonged use, nonsusceptible organisms, including fungi, may overgrow; treat superinfection appropriately.

Adverse Reactions: Hypersensitivity, including fatal anaphylaxis. Nausea, vomiting, epigastric distress, diarrhea, and black, hairy tongue. Skin eruptions, urticaria, reactions resembling serum sickness (including chills, edema, arthralgia, prostration), laryngeal edema, fever, and eosinophilia. Infrequent hemolytic anemia, leukopenia, thrombocytopenia, neuropathy, and nephropathy, usually with high doses of parenteral penicillin. [102175]

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The effectiveness of Valium (diazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral form adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling, and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status.

Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals under careful surveillance because of predisposition to habituation/dependence. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, have been observed in patients during and after Valium (diazepam) therapy and are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

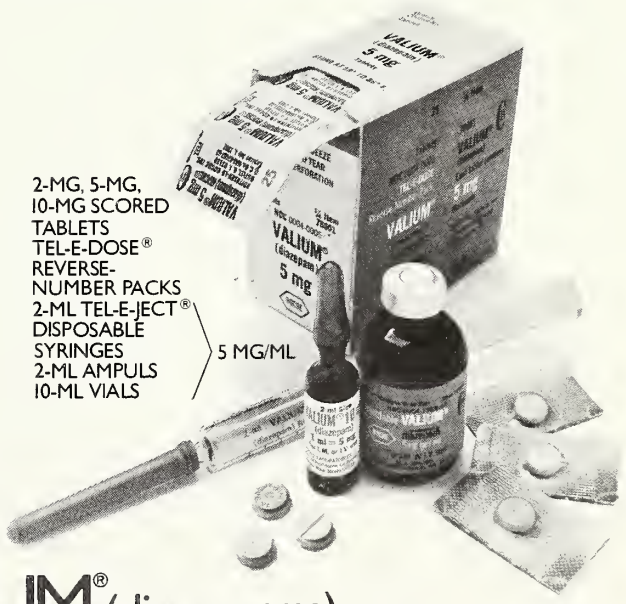
In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure, employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension, caffeine and sodium benzoate for CNS-depressive effects. Dialysis is of limited value.

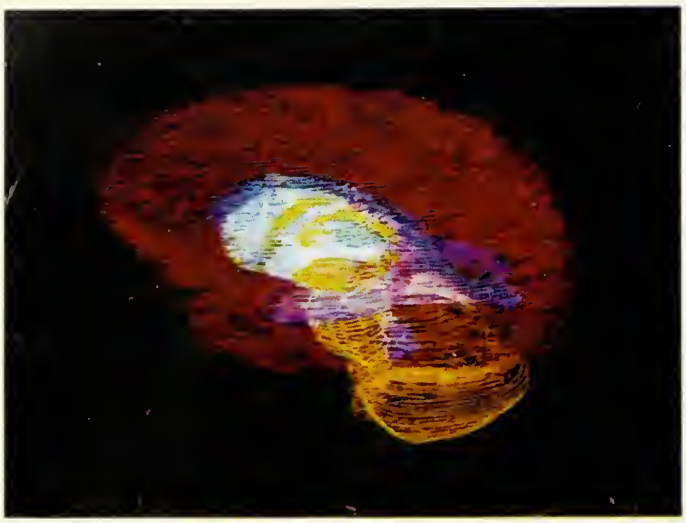
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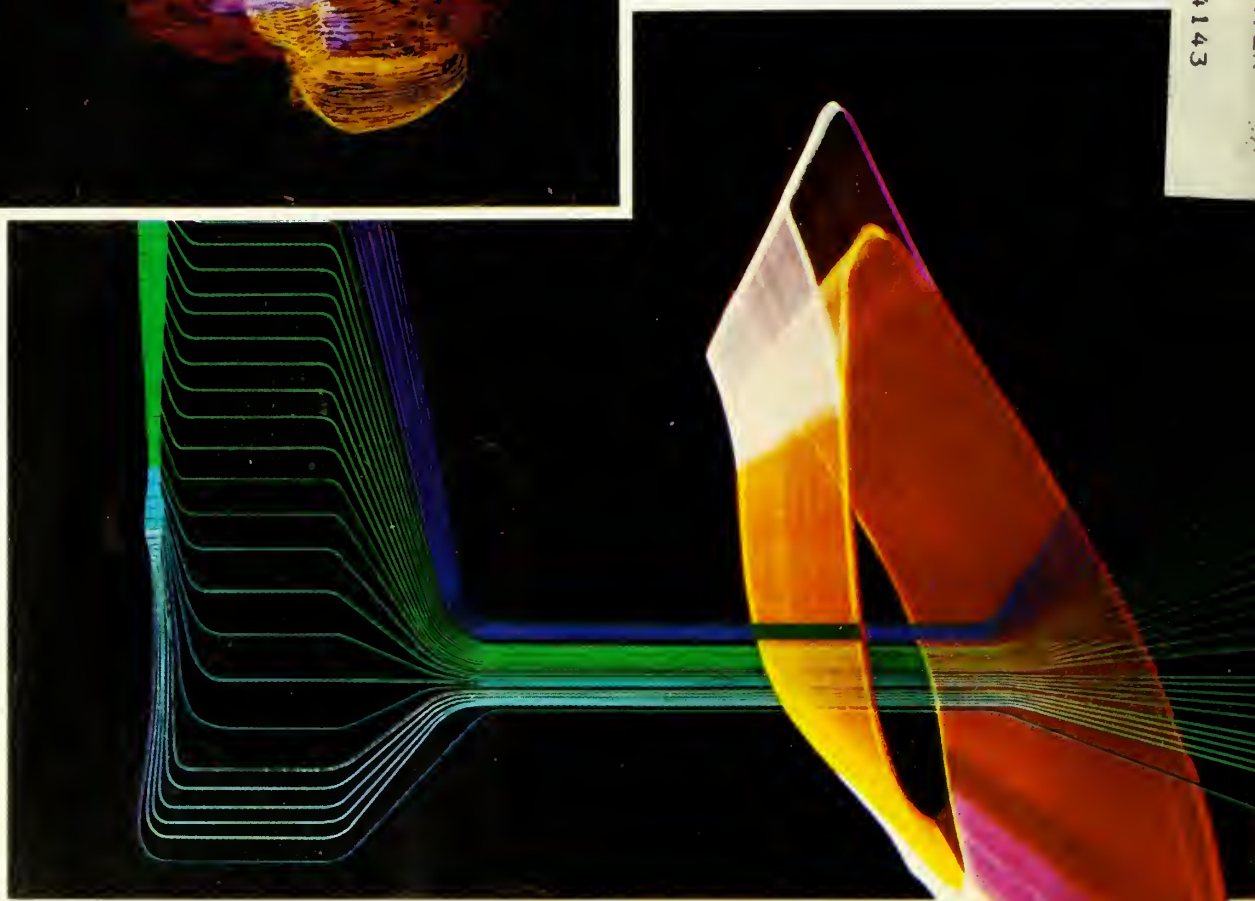
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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KANSAS MEDICAL SOCIETY

ANNUAL MEETING
May 2-4, 1980

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Friday, May 2	Infectious Diseases Symposium AMAERF Auction
Saturday, May 3	Past Presidents Breakfast First House of Delegates Reference Committee KU Alumni Reception Annual Presidents Banquet
Sunday, May 4	Second House of Delegates Council Luncheon, Meeting

PRE-CONVENTION WORKSHOPS

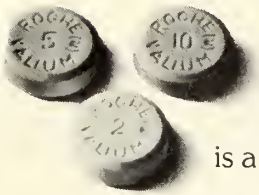
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6.5 hours CME Category I — Physicians and Spouses
Financial Planning for the Medical Family
Investments — Insurance — Estate Planning

Impairment vs Satisfaction
The Physician's Work, Marriage, and Family
1:00-5:00 PM
4 hours CME Category I — Physicians and Spouses

(To be held at Shawnee Mission Medical Center, 74th and Grandview; dinner will follow.)

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Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

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a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

ROCHE

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Division of Hoffmann-La Roche Inc.
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DISCIPLINARY ACTIONS AGAINST PHYSICIANS

In its latest survey of state medical licensing boards, the American Medical Association has charted nearly an 85 per cent increase in the number of complaints against physicians that are being investigated. The increase in actual disciplinary actions against physicians — from 685 in 1977 to 1,476 in 1978 — is part of an upward trend that was first noted by the AMA in 1971.

The increase in investigations by state boards, from 3,662 in 1977 to 6,801 the following year, can be attributed in part to the growing legal immunity offered to those who report offenses.

Fifty-seven of 60 state medical boards were surveyed for the disciplinary measures they took against physicians during 1978 (there are more than 50 state boards since some have separate osteopathic boards).

In the past, according to Lowell H. Steen, M.D., Chairman of the AMA Board of Trustees, "physicians reporting colleagues to disciplinary boards exposed themselves to the possibility of a libel suit. As more states protect people who, in good faith, report to state boards, the number of disciplinary actions also increases."

At present, 20 states have enacted all or part of the AMA's model legislation on medical discipline. (Medical discipline is one of several topics covered by AMA model state legislation. Proposed legislative language is provided as a guideline for individual states to use in drafting their own legislation.)

Some of the provisions adopted by various states require:

** that physicians report evidence of professional malpractice to the state disciplinary board;
** that medical societies report instances of unprofessional conduct;
** that health care facilities report disciplinary actions by the facility;
** that insurers report malpractice awards or settlements.

The increase in disciplinary actions taken against physicians can be attributed, in part, to the addition of more and varied forms of discipline. In addition to compiling the figures in each state for the four disciplinary measures surveyed in 1977 (revocation of medical license, suspension of narcotics permit, censure, and denial of license reciprocity), the 1978 survey also counted: voluntary surrender, suspension or probation of medical license; revocation of narcotics permit; reprimand and censure; physicians required to submit to care or counseling; physicians whose practices must be supervised; and those who need continuing medical education courses.

In noting that disciplinary boards are able to take more flexible actions, Dr. Steen said, "By giving the licensing boards something more than the drastic measure of license revocation, more appropriate and more effective discipline will result."

These wide ranging choices of action have come about largely from enactment of legislation patterned after the AMA's model state legislation.

The AMA study shows that while 216 medical licenses were revoked in 1978, an additional 302 were suspended, voluntarily surrendered, or put on probation. Reprimand and censure was brought against 268 physicians and 151 had their controlled substance licenses (drug permits) revoked or suspended.

While the AMA has provided model legislation as a guideline for individual states to consider, the actual disciplinary actions described here are taken by state medical licensing boards — not by the AMA or local medical societies. Organized medicine does not have the power to revoke or otherwise affect a physician's license.

In addition to its medical discipline model bills, the AMA has suggested legislation to identify and aid the physically or mentally impaired physician who suffers from alcoholism, drug abuse, or deterioration through aging. Thirty-one states, including Kansas, have adopted all or part of the AMA Disabled Physician Act. Further, there is increased activity by the AMA and 40 of its state medical societies to provide programs to help the impaired physician.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society met on Saturday, October 13, 1979, at the Ramada Inn, Manhattan, and submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President Elect

Herman W. Hiesterman, M.D., Quinter. Born in 1923. Graduated from the University of Kansas School of Medicine in 1951. Is in Family Practice. Is currently serving as First Vice President.

First Vice President

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Is now serving as Second Vice President and AMA Alternate Delegate.

Second Vice President

Richard L. Brownrigg, M.D., Dodge City. Born in 1935. Graduated from the University of Kansas School of Medicine in 1961. Practices Urology. Is currently serving as Councilor for District 15.

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is now Chairman, Commission on Education, and AMA Alternate Delegate.

Roger D. Warren, M.D., Hanover. Born in 1931. Graduated from the University of Kansas School of Medicine in 1957. Practices General Surgery. Is currently serving as Councilor for District 1.

Constitutional Secretary

Jack R. Cooper, M.D., Shawnee Mission. Born in 1917. Graduated from Ohio State University School of Medicine in 1943. Practices Neurosurgery. Is currently serving as Secretary.

Treasurer

William K. Walker, M.D., Sedan. Born in 1928. Graduated from the University of Kansas School of Medicine in 1945. Is in Family Practice. Is now serving as Treasurer.

Speaker

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and Speaker of the KMS House of Delegates.

Vice Speaker

John O. Yulich, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1959. Is in Family Practice. Now serves as Vice Speaker of the KMS House of Delegates.

AMA Delegate

Alex Scott, M.D., Junction City. Born in 1923. Graduated from the University of Wisconsin Medical School in 1948. Is in Family Practice. Is serving as AMA Delegate.

AMA Alternate Delegate

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Is currently serving as Second Vice President and AMA Alternate Delegate.

Quinamm™

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

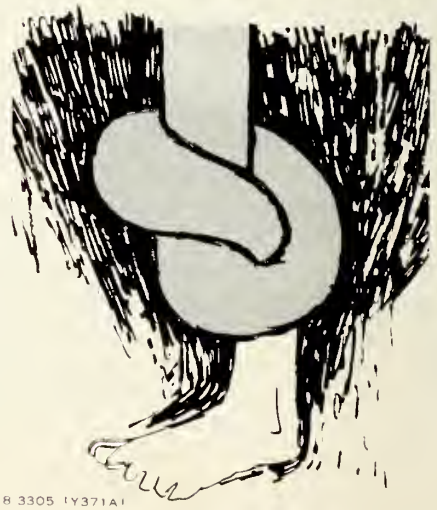
U.S. Patent 2,985,558

Merrell

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Cincinnati, Ohio 45215, U.S.A.

Licensor of Merrell®



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Quinamm™

each tablet contains quinine sulfate 260 mg., aminophylline 195 mg.

specific therapy for painful night leg cramps

Nocturnal recumbency leg muscle cramping is frequently an unwelcome bedfellow for many patients—especially those with arthritis, diabetes or peripheral vascular disease... consider Quinamm... simple, convenient dosage—usually just one tablet at bedtime... can provide restful, welcome sleep without night leg cramps.

See opposite page for prescribing information.



Tagamet®

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cimetidine

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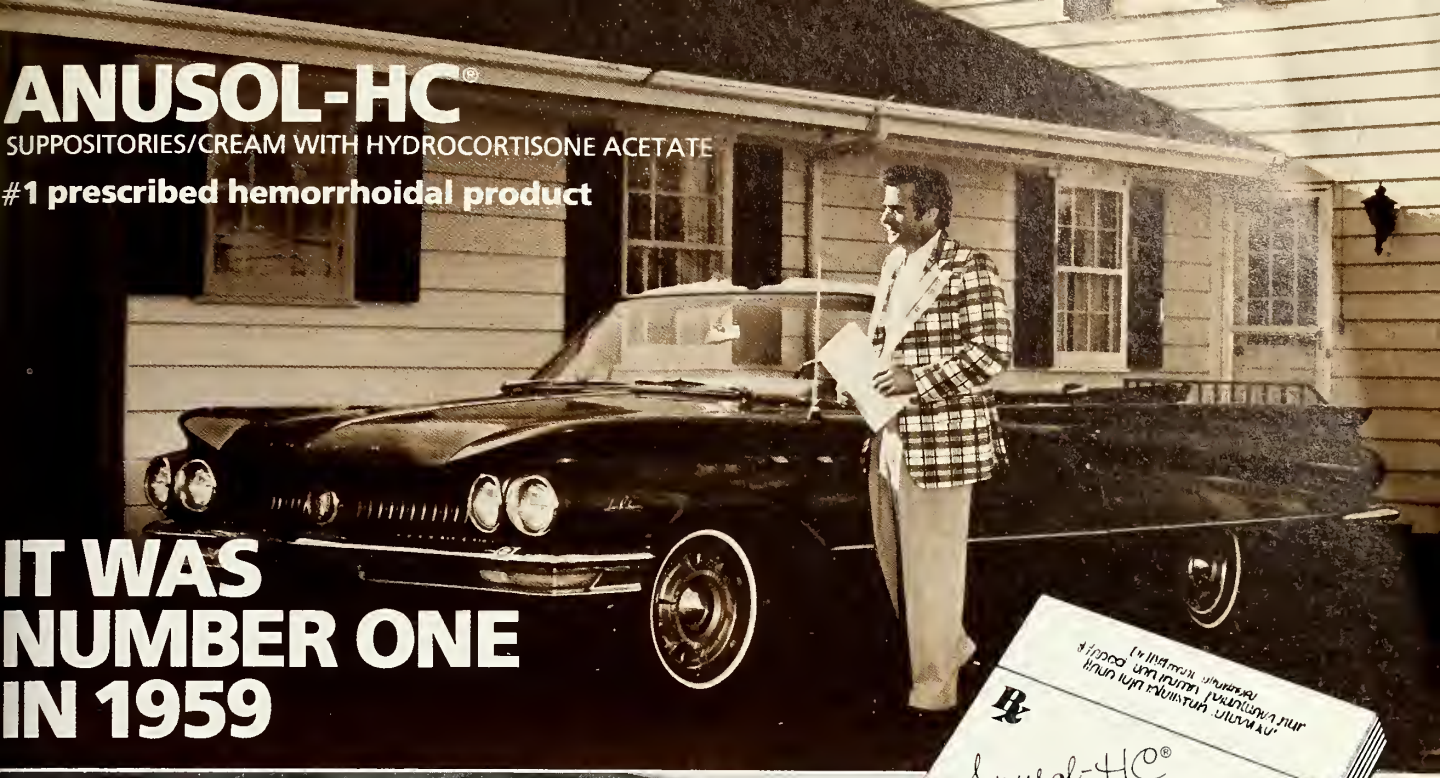
Pale green 300 mg. tablets
in bottles of 100 and Single Unit Packages of 100
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Injection, 300 mg./2 ml.,
in single-dose vials
and in 8 ml. multiple-dose vials,
both in packages of 10.

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ANUSOL-HC®

SUPPOSITORIES/CREAM WITH HYDROCORTISONE ACETATE

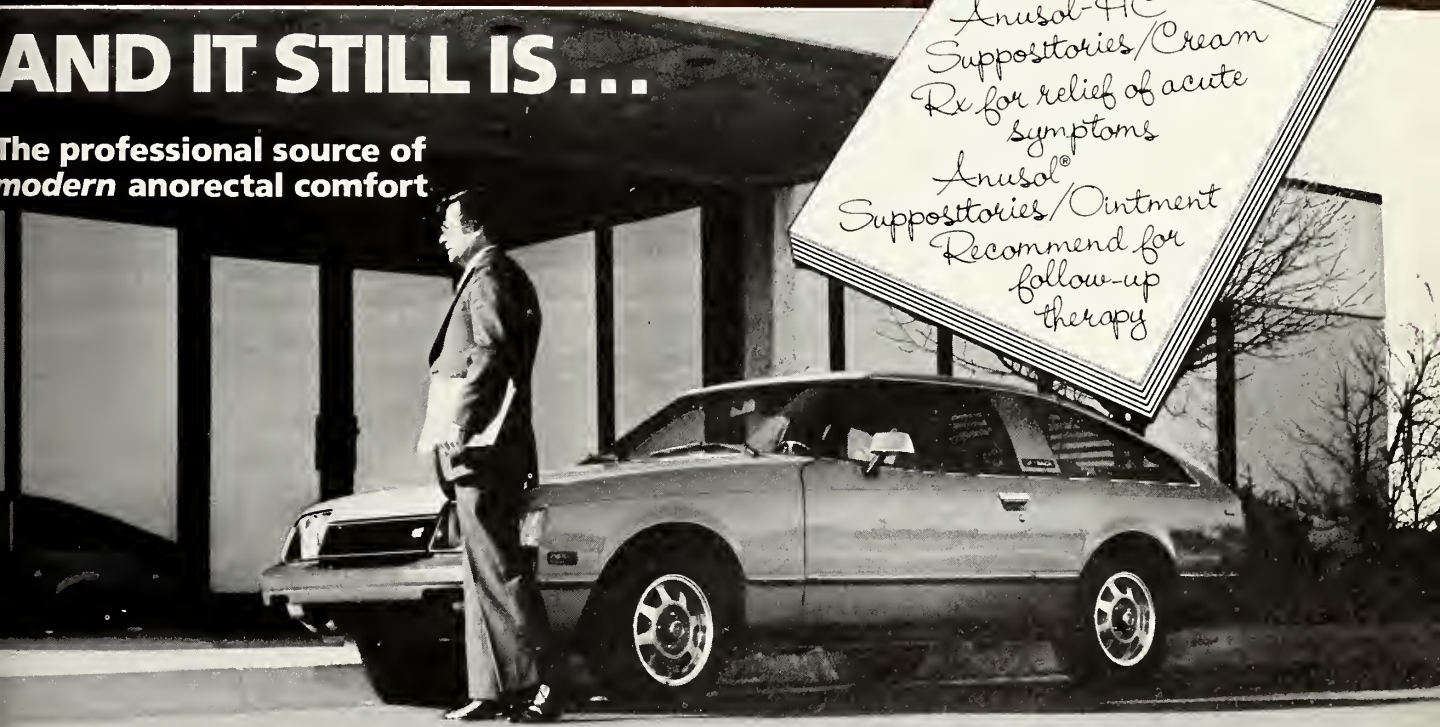
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IT WAS NUMBER ONE IN 1959

AND IT STILL IS...

The professional source of *modern* anorectal comfort



ANUSOL-HC® SUPPOSITORIES
Hemorrhoidal Suppositories
ANUSOL-HC® CREAM
Rectal Cream with Hydrocortisone Acetate
Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

Store between 59°-86° F (15°-30° C).

Full information is available on request.

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Medical HISTORY

Rambling Recollections About the "Postgraduate Department" — Now the Division of Continuing Education of the College of Health Sciences

JESSE D. RISING, M.D., *Kansas City, Kansas*

SPORADIC EFFORTS at continuing education were, for many years, conducted by the School of Medicine in cooperation with the Division of Extension at Lawrence. These were considered to be fairly successful, but were carried on in a desultory manner and, by current standards, neither well publicized nor well attended. It was not until after World War II, when physicians who had been in military service began returning, that there was a statewide outcry from the profession for continuing education (then called "postgraduate" or "post-graduate" education) to bring them up-to-date in the advances in medical practice that had occurred during their absence. This had an obvious attraction for the administration of the School of Medicine.

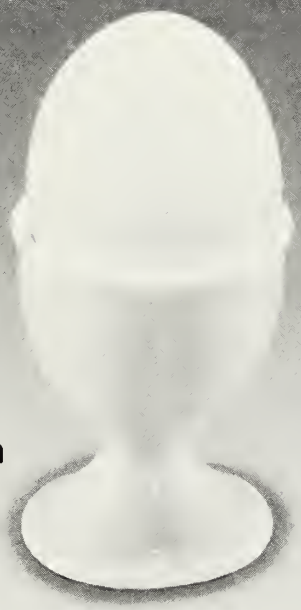
The Kansas Medical Society formed a committee on postgraduate education, which was to be advisory to the School of Medicine; and the school officially formed a Department of Postgraduate Medical Education. Edward H. Hashinger, M.D., was appointed chairman of the department in 1946. The department had no faculty of its own, but it borrowed Harold Ingham from the Extension Division in Lawrence to be "Director of the Extension Service in Medicine." It later became obvious that it would not be practical for Mr. Ingham to maintain one hundred per cent allegiance both to the Dean of the School of Medicine and to the Dean of the Extension, so his appointment was changed to Executive Director of the Department of Postgraduate Medical Education. In the beginning, he operated fairly independently with Dr. Hashinger heading up a Joint Advisory Committee in Continuing Education (composed of the members of the KMS committee and the School of Medicine committee on postgraduate programs) which developed programs and curricula. It was after the department began to attract a larger enrollment that Mr. Ingham's joint allegiance became intolerable, and Dr. Clarke Wescoe (who had become dean in 1952), persuaded Chancellor Murphy (the previous dean who had been instrumental in forming the Department of Postgraduate Medical Education), to transfer Mr. Ingham to the School of Medicine exclusively. It must be admitted that Mr. Ingham was not completely happy with this change since it decreased his independence to a considerable degree.

The original staff of the department consisted of Dr. Hashinger (a part-time chairman who actually spent most of his time in private practice and as a volunteer teacher of medical students); Mr. Ingham, executive director; and one or two secretaries. There being no field representatives, it fell to Mr. Ingham to conduct one of the circuits, and he chose the western circuit. The eastern circuit was staffed by Mr. Herbert Weatherby whose primary job was Registrar of the School of Medicine! There were no state cars at the Medical Center so each used his own vehicle and presumably charged mileage to the state budget, but it is not at all clear what the situation was. For one thing, Mr. Ingham maintained what he referred to as a "rotating fund" in Lawrence. Using this fund eliminated a bit of red tape and delay and permitted some economies. This was not discovered by the dean for nearly ten years when he was required to turn the money over to the state treasury. There has never been any hint that the "rotating fund" was

(Continued on page 156)

Ed Note: Dr. Rising has been with the Division of Continuing Education since 1953, and has served as Associate Editor of this *Journal* since 1968. In this capacity, he has been instrumental in developing and compiling the Annual UKSM-KC issue. Our best wishes accompany Dr. Rising as he leaves the Division.

IN LESS TIME THAN IT TAKES TO BOIL THIS EGG WE CAN GIVE YOU A FULL ECG INTERPRETED READOUT



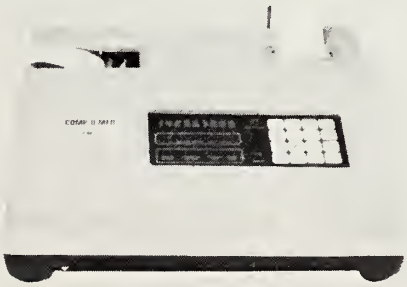
The first fully-automated, computer assisted ECG terminal designed for office use can produce a mounted trace and interpretation in less than five minutes.

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RHEUMATOLOGY TRAINEESHIP

Where: University of Kansas Medical Center

When: May 14 thru 17, 1980

For: Physicians practicing in Kansas

Purpose: To assist the practicing physician in the diagnosis and treatment of rheumatic diseases and to help him fulfill continuing education requirements.

Continuing Education Credit Requested: 20 hrs. Category I

Cost: Registration fee paid by Kansas Chapter, Arthritis Foundation. Physician responsible for meals and lodging.

Sponsored by: Kansas Chapter, Arthritis Foundation, in cooperation with Continuing Education Division, University of Kansas Medical School

RHEUMATOLOGY SYMPOSIUM

"Common Problems in Musculoskeletal Disease"

Time: 8:30 p.m.

Date: Friday, April 18, 1980

Place: Santa Fe Room
Community College
Dodge City, Kansas

Course Credit: Course is approved for 6 hours — Category I, CME credit. As an organization accredited for continuing medical education, the Kansas Chapter, Arthritis Foundation certifies that these continuing medical education offerings meet the criteria for category I credit of the Physician's Recognition Award of the American Medical Association, provided they are completed as designed.

Application made for American Academy of Family Physician CME Credit.

Application made for American Osteopathic Association, 6 hours category I, CME Credit.

Fee: $25.00

FUTURE SYMPOSIUM

"Rheumatic Diseases — 1980 Update"

Date: Friday, November 14, 1980

Place: Wichita, Kansas

Put these dates on your calendar.

For more information write to:

Charles Blackwell, Executive Director
Kansas Chapter, Arthritis Foundation
1602 E. Waterman
Wichita, Kansas 67211

NEW
PRODUCT
INFORMATION

clinical significance



of constipation

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Constipation acute or chronic

Constipation may be caused by conditions affecting the filling and emptying of the rectum.

Inadequate filling

Interference in propulsive contractions

Impairment of smooth muscle contractility

Obstruction of the lumen





Inadequate emptying

Interference in the stimulation of the defecation reflex

An additional complication

Self treatment—use and abuse of laxatives

Treatment of underlying disorders is critical... Relief of constipation is essential

Perdiem™...distinctive!

A unique blend of natural vegetable derivatives For comfortable and safe relief of constipation

Psyllium

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- Strengthens stimulus to defecate by increasing indigestible residue
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Senna

- Produces mild peristaltic stimulotion
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A unique granular formulation

- No mixing ar chewing
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- Helps break cothartic habituation
- Helps establish narmal defecotary reflexes and regular bawel rhythm

John Moerz, M.D.
Medical Director
W. H. Rorer, Inc.
Fart Woshingtan, PA 19034

Dear Dr. Moerz:

Yes, I would like to receive a supply of Perdiem™ starter samples for my patients.

Dr. _____

Address _____

City _____

State _____

Zip _____

Specialty _____

SJ-312

New Perdiem™

Prescribing Information

ACTIONS: Perdiem™, with its gentle action, does not produce disagreeable side effects. The vegetable mucilages of Perdiem™ soften the stool and provide pain-free evacuation of the bowel. Perdiem™ is effective as an aid to elimination for the hemorrhoid or fissure patient prior to and following surgery.

COMPOSITION: Natural vegetable derivatives: A unique blend of psyllium and senna (Plantago Hydrocolloid with Cassia Pod Concentrate).

INDICATION: For relief of constipation.

PATIENT WARNING: Should not be used in the presence of undiagnosed abdominal pain. Frequent or prolonged use without the direction of a physician is not recommended. Such use may lead to laxative dependence.

DIRECTIONS FOR USE—ADULTS: Before breakfast and after the evening meal, one to two rounded teaspoonfuls of Perdiem™ granules should be placed in the mouth and swallowed with a full glass of warm or cold beverage. Perdiem™ granules should not be chewed. After Perdiem™ takes effect (usually after 24 hours, but possibly not before 36-48 hours): reduce the morning and evening doses to one rounded teaspoonful. Subsequent doses should be adjusted after adequate laxation is obtained.

IN OBSTINATE CASES: Perdiem™ may be taken more frequently, up to two rounded teaspoonfuls every six hours.

FOR PATIENTS HABITUATED TO STRONG PURGATIVES: Two rounded teaspoonfuls of Perdiem™ in the morning and evening may be required along with half the usual dose of the purgative being used. The purgative should be discontinued as soon as possible and the dosage of Perdiem™ granules reduced when and if bowel tone shows lessened laxative dependence.

FOR COLOSTOMY PATIENTS: To ensure formed stools, give one to two rounded teaspoonfuls of Perdiem™ in the evening with warm liquid.

DURING PREGNANCY: Give one to two rounded teaspoonfuls each evening.

FOR CLINICAL REGULATION: For patients confined to bed, for those of inactive habits, and in the presence of cardiovascular disease where straining must be avoided, one rounded teaspoonful of Perdiem™ taken once or twice daily will provide regular bowel habits. Take with a full glass of water or beverage.

FOR CHILDREN: From age 7—11 years, give one rounded teaspoonful one to two times daily. From age 12 and older, give adult dosage.

NOTE: It is extremely important that Perdiem™ should be taken with a plentiful supply of liquid.

HOW SUPPLIED: Granules; 100 gram (3.5 oz.) and 250 gram (8.8 oz.) canisters.



Made in West Germany

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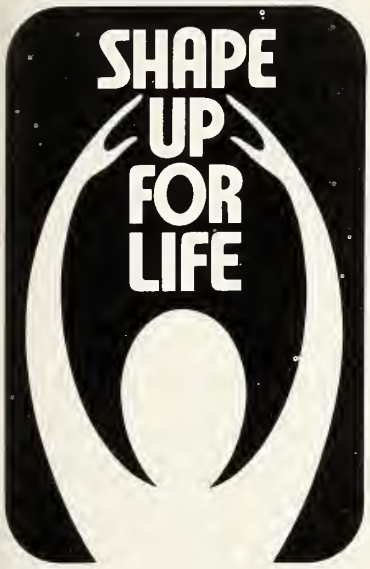
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American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

Auxiliary News

"Time and tide wait for no man."

These words, penned by John Heywood, bring upon me the realization that another Auxiliary year is fast approaching its completion. There were times, I must admit, when I wished it would speed its course, but at this moment, time is of the essence!

With the beauty of springtime come also housecleaning chores and an assessment of the past year's gains and losses. So it is with the Auxiliary on both the county and state levels. Reports are being readied, membership lists updated, and projects completed. Meantime, we are also concentrating on fulfilling goals set nearly a year ago. As we do this, Evelyn Huff and I continue to visit Auxiliary chapters across the state. In January we attended the Sedgwick County Auxiliary, which is under the expert guidance of Mrs. John Rempel. The following day we journeyed on to Wellington to meet with Mrs. Joel Weigand, president, and the South Central Tri-Counties Auxiliary. They are a small group and have the problem of distance, but I sensed a renewed interest among the members in attendance. In February I visited the Labette County Auxiliary, who meet every month with their husbands. Mrs. Ned Gorrell is their enthusiastic president. From Parsons, I traveled on to Caney, where the Southeast Counties Auxiliary met at the home of our First Vice President, Mrs. Robert Moore. Mrs. William Campbell conducted the meeting in the absence of their president, Mrs. William Walker. I am looking forward to yet visiting with the Reno County Auxiliary, under the leadership of Mrs. Don Brada, and the Butler-Greenwood Auxiliary, who boast Mrs. James Chapman as their president. We will wind up the year's meetings with the Wyandotte and Johnson County auxilians as they put their forces together to finalize convention plans.

Yes, convention time is just around the corner, and we are very excited about the many new events that will take place. Among the "firsts" will be a participation clinic to emphasize our theme, "Shape Up For Life." Secondly, our eyes are especially focusing on the First Annual (we hope!) AMAERF auction. Also, we are extremely fortunate and honored to have Mrs. Ben Johnson, Jr., President of the American Medical Association Auxiliary, as our guest throughout the convention. Please begin *now* to make plans to attend along with your spouse.

Dee (Mrs. Wilbur G.) Cauble of Wichita was named Kansan of the Year for 1980 during the annual banquet of the Native Sons and Daughters of Kansas, January 25, in Topeka. This honor is bestowed yearly on an outstanding Kansas citizen during the Kansas Day festivities, and Dee was recognized for her long and tireless service through several world-wide health organizations. We are pleased that this dedicated KMSA member has received public recognition for her outstanding contributions.

Please check to see if your spouse's dues have been paid. We are concerned that we will lose our delegate count of past years at both state and national convention if the present trend prevails.

Thank you for your support and interest this past year. Remember, it is our goal to support and aid your role as physician and spouse in our homes and communities by promoting health education through "healthful lifestyles" — our theme this year.

Sincerely,
Kathy Wedel
President
Kansas Medical Society Auxiliary

An open letter to Physicians

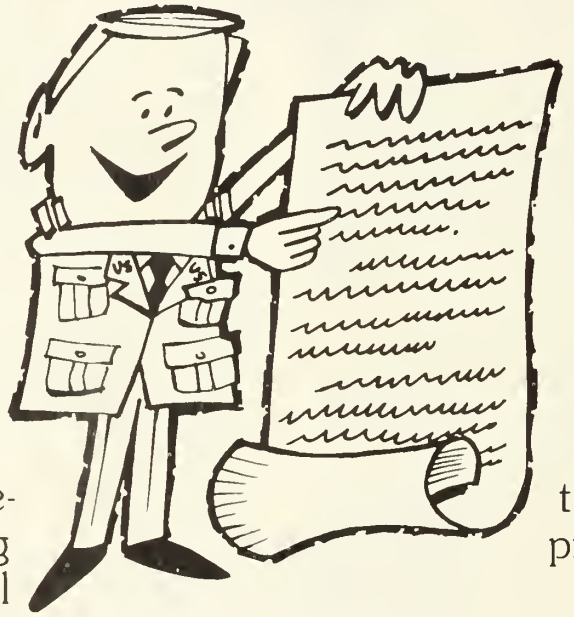
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Thirty-Fourth Annual University of Kansas School of Medicine Issue

The Dean's Letter — 1980

The University of Kansas Medical Center

David Waxman, M.D.,* *Kansas City, Kansas*

ONCE AGAIN, it's my privilege to address the readers of the *Journal of the Kansas Medical Society*. One year ago, I described the status of several new and continuing programs at the University of Kansas College of Health Sciences and Hospital. I will take advantage of this opportunity to bring you up-to-date on those activities and to share with you some recent developments.

Enrollment at the School of Medicine will soon reach nearly 700 students. We continue to approach the 800 mark with each additional entering class of 200 students. Approximately 75 of these future physicians are completing their clinical clerkships at the University of Kansas School of Medicine in Wichita, with the number to increase to 100 in 1981. The Kansas Legislature has identified the E. B. Allen Hospital as the permanent home of the Wichita Branch and has designated 1.3 million dollars for renovation and improvements for the building. Enrollments in our other divisions — the schools of nursing, allied health, and graduate programs in the biomedical sciences — have continued to rise. There are now over 2300 students enrolled in educational programs at the University of Kansas College of Health Sciences.

The new Bell Memorial Hospital has opened to patient care. It has allowed us to significantly expand the quality and range of services to our patients and their families, while at the same time providing a superb environment in which to train future physicians, nurses, and allied health personnel.

The new Radiation Therapy Center with its 40 million, 20 million, and 6 million electron volt linear accelerators represents a quantum leap forward in our ability to treat cancer patients with the most sophisticated means available. A new parking facility has gone a long way toward improving access to the KU Medical Center for patients and other visitors. Finally, progress continues in the planning for a new Health Sciences Library at the Kansas City campus.

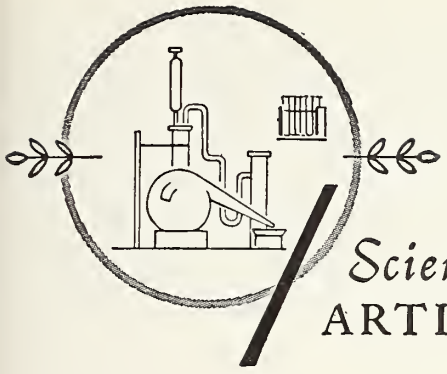
During the past year there were important acknowledgements of the quality of our educational and hospital programs. The University of Kansas received the highest possible recognition from three major accrediting bodies — full accreditation for the School of Medicine (7 years); full accreditation for the School of Nursing (8 years), and full accreditation for the University Hospital (2 years). These are the goals of excellence to which all university medical centers aspire; they signal the high achievements of our faculty, students, staff, and administrators.

Since it was formed less than three years ago, the Division of Health Care Outreach has been increasingly involved in community based educational programs for health professionals, visits by students to community physicians, *locum tenens*, placement of "off-year" medical graduates, assistance to communities recruiting physicians, and in several other areas. Our faculty, residents, and students have participated in more off-campus programs than in any comparable period in our history. Medical faculty conduct problem-oriented, continuing education programs in more than two dozen Kansas communities, with physicians from various other communities participating. Continuing education programs for community hospital and other nurses take place in nearly three dozen communities across the state. Graduate nursing courses are conducted in six locations in addition to Kansas City.

Regional coordination of our Outreach programs and the development of new programs in response to community requests are the responsibilities of our regional health education centers. Our center in Chanute continues to provide a growing number of educational and community services in a 15-county region of southeast Kansas. The Division of Health Care Outreach at the Wichita Branch serves a multicounty area in southcentral Kansas, and a new center has been established in Hays that involves a twenty-seven county area in northwest Kansas. Other centers in Garden City and Topeka are being developed to serve southwest and northeast regions of the state respectively. All of these regional programs receive guidance and direction from advisory committees that include health professionals, hospital adminis-

(Continued on page 114)

* The Executive Vice Chancellor, University of Kansas School of Medicine, Kansas City, KS 66103.



Spasticity

Current Concepts of Management

GEORGE VARGHESE, M.D.* *and* JOHN B. REDFORD, M.D.,† *Kansas City, Kansas*

Some degree of spasticity may be helpful to central nervous system disorders. However, severe spasticity presents therapeutic problems that necessitate appropriate management. The mechanics of spasticity and various regimens of treatment are discussed.

SPASTICITY is known to be a therapeutic problem in many patients with upper motor neuron lesions. With the increase in incidence of head injuries and spinal cord injuries and better care available to these patients, management of spastic patients will present an increasing therapeutic challenge to many physicians. In addition other central nervous system lesions — such as cerebrovascular accidents and multiple sclerosis — which also manifest spasticity are not declining. Spasticity may be the biggest problem in daily living for many patients.

Spasticity is a clinical condition wherein the striated muscle shows increased tonicity — when the muscle is manipulated, increased resistance to stretch is felt. Spasticity is to some extent helpful for the patient. The constant muscle contraction stimulates the periosteum and retards the osteoporotic activity. It decreases the possibility of thrombophlebitis. Spastic lower limb and trunk muscles also help to improve balance and ambulation in many patients. Unfortunately, the spasticity is frequently so severe that it interferes with exercise programs, activities of daily living, bed and wheelchair transfer activity, and balance. It may lead to the formation of joint contractures. The management of spasticity is often frustrating to both patient and physician.

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Pathophysiology

Before discussing the pathophysiology of spasticity, certain anatomic and physiological features of spinal reflex arc must be reviewed. The muscle spindles comprised of the intrafusal muscle fibers form the receptor organs to initiate spinal reflexes. Muscle spindles lie within the skeletal muscle parallel to the larger extrafusal fibers. When the tendon is tapped or muscle is stretched, muscle spindles are stimulated. The afferent nerve fibers from these structures end in the gray matter of the spinal cord where they synapse with the alpha motor neurons *(Figure 1)*. Efferent impulses from the alpha motor neurons produce contraction of the muscle itself. The anterior horn of the spinal cord also contains the gamma motor neurons. These gamma neurons innervate the muscle spindle and are under the facilitory and inhibitory

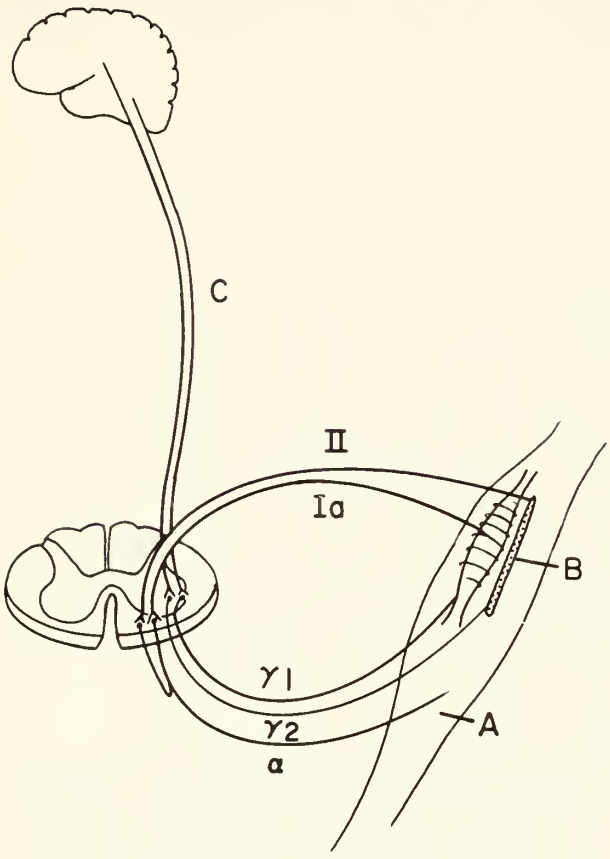


Figure 1. Spinal reflex arc. A. Extrafusal muscle fibers. B. Muscle spindles. C. Descending fibers to spinal reflex arc. Ia and II Afferent fibers from muscle spindle to spinal neuron. γ and γ Efferent fibers to muscle spindle. α — Efferent fibers to extrafusal muscle fibers.

influences from the supraspinal centers.[1, 2] The afferent impulses coming from the muscle spindles will synapse with the motor neurons going into the same muscle and the synergistic muscles. These impulses are excitatory in nature. Other afferent impulses which are inhibitory in nature go to the alpha neuron of the antagonistic muscle by way of interneurons.[1] For example: The afferent impulses from the muscle spindle from the biceps will excite the alpha neuron supplying the biceps and brachialis and inhibit the alpha neuron supplying the triceps and anconeus. As a result, there is contraction of the biceps and brachialis and relaxation of the anconeus and triceps producing smooth flexion of the elbow joint. These spinal reflexes are constantly modulated by the supraspinal centers. Supraspinal influences are either inhibitory or facilitory. The descending pathways involved in the modulation are corticospinal, rubrospinal, reticulospinal, and vestibulospinal.[1] As a consequence of a destructive lesion of the cortex (such as a stroke) or all the spinal tracts (such as a spinal cord injury), the spinal reflexes are deprived of supraspinal control. When the muscle spindle is stimulated, the afferent stimulus reaches the spinal cord which in turn stimulates alpha motor neuron to produce contraction of the extrafusal fibers. Since there is no inhibitory influence to modulate the contraction, the lesion will allow a sustained contraction which the clinician calls spasticity.

Clinical Features

Spasticity is characterized by an increase in the muscle tone when the muscle is stimulated or manipulated. The presence of spasticity always implies an underlying neurological disorder. This has to be differentiated from a local muscle spasm secondary to trauma or contractures secondary to prolonged immobilization and positioning of the joints in a shortened position. Spasticity has to be differentiated also from the rigidity noted in parkinsonism. Spasticity is associated with hyper-reflexia and clonus which are not seen in parkinsonism. If the muscle is stretched, the degree of spasticity depends on the speed of stretch of the muscle. The spastic muscle may relax toward the end of the passive movement and this sudden give-away is called "the clasped knife" phenomenon. In parkinsonism there is a gradual sustained resistance to the passive stretching movement termed rigidity which is independent of the speed of stretch. This kind of resistance is often called "cog-wheel" rigidity as tremor is often noted along with the rigidity.

Various central nervous system lesions can manifest spasticity and the following common conditions are associated with it:

1. Cerebrovascular disorders involving the cortex or the brain stem;
2. Head injuries resulting in cortical or brain stem damage;
3. Spinal cord injuries. Any lesion, whether complete or incomplete, either traumatic or otherwise, above the conus medullaris;
4. Demyelinating disorders such as multiple sclerosis, transverse myelitis;
5. Amyotrophic lateral sclerosis;
6. Cerebral palsy;
7. Metabolic disorders including such inherited disorders such as Tay-Sachs disease, metachromatic leukodystrophy; and
8. Vitamin B_{12} deficiency (subacute combined degeneration).

Spasticity varies considerably from individual to individual with the same disorder. Spinal cord disor-

ders usually have more spasticity than cortical or brain stem disorders — possibly because in a spinal cord injury, all higher pathways influencing the spinal reflexes are interrupted. The severity of the spasticity fluctuates in the same individual and there is a distinct diurinal variation in its degree. Associated conditions, such as urinary tract infections, urinary calculi, decubitus ulcers, and fecal impaction can lead to temporary increase in spasticity. Emotional stress commonly produces an increase in spasticity.

Clinical Management

Spasticity as noted earlier is often beneficial to the patient. However, when it interferes with day-to-day function — for example, in dressing and in ambulation — it must be treated. Evaluation of its effects before prescribing treatment is of prime importance. Some of these patients demonstrate severe flexor spasm by drawing up of both lower extremities almost into the fetal position whenever the muscle is stimulated. This is a frustrating condition and definitely requires treatment. Some patients demonstrate extensor patterns when they try to transfer or ambulate. This extensor pattern can cause them to lose their balance and fall. Patients may complain that they are awakened from sleep many times because of the flexor spasm. Observation of the patient while in bed and during various activities will demonstrate some of these increased spastic responses. With the new medical treatments available, it is possible to ease some of these annoying effects of spasticity even though they cannot be completely abolished.

General Measures

When conditions exist that can aggravate spasticity, treatment of this is the first step. Proper prevention and treatment of urinary tract infections, decubitus ulcers, and fecal impaction can prevent or relieve the spasticity to a certain extent. Whenever a patient complains that he is experiencing increased spasticity, he should be checked for any of the above mentioned conditions. Many times careful physical examination or some simple laboratory investigations will enable the physician to make the diagnosis of the aggravating factor or condition. Appropriate treatment of this condition itself can diminish the spasticity without resorting to expensive and complicated anti-spastic medications or surgery.

Medical Treatment

The search for an effective drug for treating spasticity has been a long and difficult one. Over the years many drugs have been evaluated for control spasticity without much success. The main problems with most of the medications were generalized effects on the brain and on even the normal muscles. Some of the drugs used earlier — such as mephenesin (Myanesin) and methocarbamol (Robaxin) — were found to be effective as skeletal muscle relaxants. These drugs have a depressant effect on polysynaptic and monosynaptic reflexes. Unfortunately, they also have a depressant action on reticular formation causing drowsiness when they are used in therapeutic doses.[3] Because of the failure of drug treatment, intrathecal injection and surgical procedures were popular in the past. Recently several drugs have been developed for control of spasticity, and surgery is not as prevalent. The drugs commonly used today for treatment of spasticity are diazepam (Valium), dantrolene sodium (Dantrium), and baclofen (Lioresal).

Diazepam (Valium) has a central effect on the limbic system and the reticular activating system inducing a calming effect. It exerts an inhibitory influence on polysynaptic reflexes in the spinal cord, and also may have some suppressive effect on the contractile property of the muscle fiber. It is fairly effective in reducing spasticity and clonus. Usual oral dosage ranges from 10-40 mg/day. Usually the patient is started on a small dosage of 5 mg 2 times/day and then gradually increased to the optimal dosage. Drowsiness is the main side effect of diazepam, especially in the early phase of treatment. Fatigue and ataxia are also noted. Occasional overdosage and habituation must be considered in patients with spinal cord injury.

Baclofen (Lioresal) is a synthetic derivative of gamma amino butyric acid which, like diazepam, has an inhibitory influence on the synaptic reflex activity in the spinal cord. Baclofen is generally recommended only for spinal cord lesions such as those caused by trauma or multiple sclerosis, and is not recommended for hemispheric lesions. The optimal dosage is 45-80 mg/day. The dosage must be titrated for each individual patient as follows: a dose of 5 mg 2 or 3 times/day is gradually increased every third or fourth day. The patient may complain of sleepiness during the early treatment phase, but beginning with a small dose and then gradually increasing the dosage usually mitigates this problem. In addition to sleepiness, side effects noted with this drug include dizziness, anorexia, and constipation.[3, 4] No significant liver, kidney, or hemapoietic effects have been reported so far.[5] Baclofen decreases the most disturbing forms of spasticity such as flexor spasm and clonus. In case the baclofen must

be discontinued for any reason, it is better to gradually decrease the dosage because abrupt termination may cause an increased spasticity or rebound phenomena.[6]

Dantrolene sodium (Dantrium) acts peripherally at the sarcoplasmic reticulum inside the muscle fiber. It probably decreases the calcium release from the T-system within the muscle fiber. The usual effective dosage is 200-400 mg orally/day. Dantrium should be started at a lower dosage of 25 mg 2 times/day and then gradually increased. In addition to drowsiness, patients may complain of weakness in the non-spastic muscles, and this is a major side effect, especially in the early phase of treatment. As liver toxicity has been reported with Dantrium, the patient's liver function should be checked before starting the treatment. It is recommended that liver function tests be repeated every two to three weeks during the initial two to three months, again in four to six months, and afterwards once every four to six months while the patient is receiving Dantrium. This drug is not recommended for patients with suspected liver disease.

Injection with Sclerosing Agents

Injection of alcohol and phenol intrathecally and also into the peripheral nerves has been used since the 1930s. Intrathecal injection of absolute alcohol completely destroys the reflex activity and produces flaccidity below the level of injection. Disadvantages of this procedure are loss of sphincter function and sexual function. Occasionally the level of lesion may even rise proximally by one or two segments. Effects of injection with phenol are relatively less permanent than with alcohol. Phenol can also be injected directly into the peripheral nerves or into the motor points of the muscle. Usually 5-7 per cent phenol in distilled water is used.[7,8] Blocking the peripheral nerve is easier than at the motor point but in a patient with intact sensation, this might destroy sensation or produce uncomfortable dysesthesia. Another consideration is that nerve injection will reduce spasticity only in the muscles supplied by that particular nerve. This is a useful procedure to reduce spasticity in a particular group of muscles. For example, if severe hip adductor spasm is resulting in a nursing and hygiene problem, the blocking of the obturator nerve will be helpful. Injecting the posterior tibial nerve in patients with severe plantar flexion spasm is hindering ambulation is another possible application. Injecting phenol into the motor points of the muscle will also reduce spasticity. As the spasticity is reduced only in that particular muscle, this process can be more selective than the nerve infiltration. Another advantage of this procedure is that it reduces spasticity without disturbing the sensation. The disadvantage of this procedure is that it is often difficult to hit the exact motor point. This is considered to be a good procedure in cerebral palsy and also following stroke because in these patients it is desirable to preserve the sensation and at the same time reduce spasticity.

Unfortunately, the effect of the phenol block lasts only 6-12 months and then gradually wears off. However, phenol block could be considered as a good therapeutic test before subjecting the patient to any neurectomy or tenotomy. For example, if the blocking of the gastrocnemius-soleus improves the gait in a patient who has a spastic equinus deformity, such a patient would be likely to benefit from tendon achilles lengthening.

Surgical Procedures

Peripheral Neurectomy and Tenotomy. Section of the peripheral nerve will completely eliminate spasticity in the muscles supplied by the nerve. This is a permanent procedure compared to the phenol nerve block, and like that procedure, the effect is only in the muscle supplied by that particular nerve. Common nerves subjected to nerve section are the obturator and the posterior tibial nerves. Sectioning of the muscle at the insertion is also used to reduce spasticity. Adductor tenotomy, for example, is often done for severe adductor spasm in cerebral palsied children.

Posterior Rhizotomy. Sectioning of the posterior root will abolish the afferent impulses coming to the reflex arc. For the lower extremities this section is done from L-2 to S-2 level and for the upper extremities from C-4 to T-1 level.[9] The results are generally good as far as spasticity is concerned. The main disadvantages of this procedure are the high frequency of recurrences of spasticity and severe sensory disturbance in the affected extremity.

Anterior Rhizotomy. Sectioning of the anterior root will completely abolish the spasticity because the efferent impulses cannot reach the muscle. Usually it is done from T-11 to S-1 level. This procedure will result in permanent paralysis and flaccidity. Any residual voluntary sphincter control also will be lost,[9] so this procedure is considered only in those patients where there is complete lack of motor and sphincter control below the lesion.

Myelotomy. Myelotomy is the most attractive neurosurgical procedure for spasticity *(Figure 2)*. In 1951, Bischoff described a technique consisting of

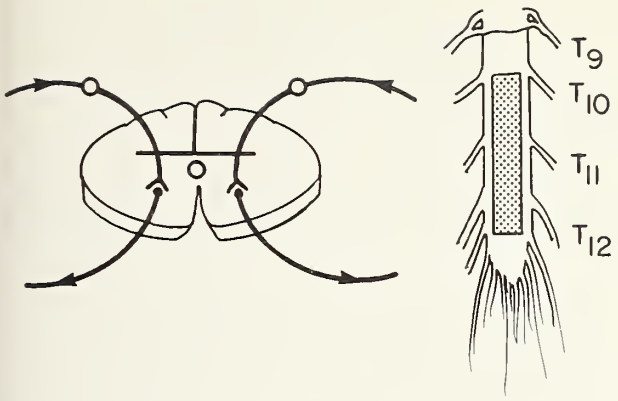


Figure 2. Schematic drawing of "T" myelotomy.

longitudinal division of the spinal cord into an anterior and posterior half from T-12 to S-1.[10] This division interrupts both monosynaptic and polysynaptic reflex arcs, at the same time preserving all long tracts. The procedure involves a laminectomy from T-10 to T-12, and division of the cord from T-11 to S-1.[9, 11] This effectively abolishes spasticity in the lower extremities. One complication of this procedure is that it may result in the loss of any residual motor or bladder function. The modification of this procedure called "T" myelotomy has been described. In this procedure dorsal midline myelotomy with a lateral extension is done to avoid abolishing any preserved bladder or motor function. Recurrence rate of spasticity after this procedure is very low.[9] It has to be noted that this procedure involves a major neurosurgical procedure and therefore must be used only in selected cases.

Physical Therapy Techniques

Several physical therapy techniques have been introduced to reduce spasticity. These techniques are based on the fact that alterations in the sensory stimulation and posture have facilitory or inhibitory influences on the muscle tone. Even though the long term effect of these techniques is not certain, the immediate effects are undeniable.[12] It is often possible to train patients to use these techniques to help themselves in functional activities such as ambulation and transfer between bed and chair. Such physical therapy techniques include local cooling, special posture exercises, and muscle stretching.

Local Cooling. Local cooling of the muscle either by cold packs or immersion in cold water is known to reduce spasticity. This is most likely due to the reduction in the threshold of stimulation of muscle spindle by local cooling. Cooling of the muscle for a period of 25-30 minutes can reduce the spasticity for as long as three to four hours. During this period a patient can go through the range of motion of the joints and so can facilitate mobility for at least a few hours. Cryotherapy also is used as a test to differentiate between alpha spasticity and gamma spasticity. In gamma spasticity there is hyperactivity of the muscle spindle and cryotherapy will diminish the inflow from the muscle spindle. This gamma spasticity is often seen following spinal cord injury. The alpha spasticity due to supraspinal factors is independent of the muscle spindle and does not respond to cryotherapy. Cooling spastic muscles can be used as a therapeutic test to check the efficacy of baclofen and Valium, as patients who respond to cryotherapy will most likely also respond to Valium and baclofen.

Posture. It has been noted by Bobath and others that positioning the body in certain ways can alter spasticity by inhibiting undesirable reflex effects. For example, flexion of the neck can reduce some of the extensor spasms noted in the lower extremities. Changes from a supine to a prone position may relieve extensor spasms. Extension and abduction of the shoulder and extension of the elbow and wrist during ambulation can reduce some of the flexor spasm noted in hemiplegic ambulation. It is also noted that facilitation of the antigravity muscles — by tapping, vibration, or some other form of sensory stimulation — improves the contraction. It is believed that with repeated attempts to initiate this contraction it is possible for the patient to learn to a certain extent to bring the movement under voluntary control. These techniques are extensively used both in cerebral palsy and in stroke patients and have been proved very helpful, especially in cerebral palsy patients.

Exercises. Maximum contraction of the extrafusal muscle fibers whenever possible can unload the muscle spindle of some of the stimulation. As a result of an activation of the golgi tendon organ within the extrafusal muscle fibers, autogenic inhibition of the muscle spindle stimulation occurs.[12] On the basis of this neurophysiological phenomenon, exercises and even electrical stimulation have been used to reduce spasticity.

Bracing and Splinting. Limiting joint movements and complete stabilizing of joints are known to reduce some of the uncontrolled or spastic movements at the joint. The constant stretch applied by the brace or the splint can facilitate some of the autogenic inhibition of muscle spindle as noted before. The short leg or below-knee brace in a hemiplegic patient

is an example of reduction of spasticity by use of a brace. With the brace preventing plantar flexion, it is often possible to reduce the spastic equinus deformity which very often inhibits the patient's ambulation. Splinting and bracing also can prevent contractures that could result from prolonged spasticity. For example, very often an hemiplegic hand can develop extreme flexion contracture of the wrist and finger joints. By positioning the hand in a functional position, contracture formation in the hand joints can be prevented.

Summary

Spasticity is noted in the majority of upper motor neuron lesions such as cerebrovascular accidents and spinal cord injuries. To a great extent, spasticity is helpful in reducing some of the complications such as thrombophlebitis and osteoporosis. However, spasticity often is so severe that it becomes frustrating both to the patient and to the physician. Recently several drugs have been introduced that could effectively reduce spasticity. Some patients do not respond to oral drug therapy and may need injections or surgical treatment. Some of the common medications used and the surgical techniques available have been reviewed.

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The Dean's Letter

(Continued from page 108)

trators, educational institutions, health planning agencies, and ordinary citizens.

To further enhance our delivery of continuing education programs geared to the needs of practicing physicians, the divisions of Health Care Outreach and Continuing Education have been merged. The reorganized Division of Health Care Outreach and Continuing Education will continue its perennial circuit courses for physicians as well as offering conferences and symposia in Kansas City and elsewhere. Battenfeld Auditorium at the UKSM campus has undergone cosmetic surgery and is now a much more attractive conference hall. Continuing education programs include book displays, poster and audiovisual exhibits, demonstrations of computerized literature searches, small group meetings, and other features to enhance the educational quality of our CE sessions.

I've tried to indicate briefly some of the achievements of the past year and some of the directions in which we're heading. In the year ahead, we will continue to improve our ongoing activities, to consolidate developing programs, and to be sensitive and responsive to the need for new efforts. Our progress depends in large measure upon open lines of communication between the University and our colleagues in the medical and other professions. I hope that we continue to have your support as we pursue our goals, and I welcome your comments, criticisms, and concerns as we move ahead with the task.

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Patient Education

Effective Methods in Primary Care Practice

NORMAN G. MARVIN, M.D.,* *Kansas City, Kansas*

Patient education can be utilized to facilitate healing, enhance the relationship between patient and health care professionals, and decrease health care costs. The procedures developed in the Family Practice Department at UKSM-KC are readily adaptable to most health care environments.

PATIENT EDUCATION may be defined as a process occurring between health care professionals and patients for their mutual benefit. Patient education in the past has been approached in various ways — mostly informal, unplanned, and on a one-to-one basis. This method may be adequate, but often opportunities for patient teaching are unrecognized and unused. This paper will demonstrate how patient education is accomplished in the Family Practice Department at UKSM with those facilities available in most practice settings.

The quality and content of patient education varies widely. Recognizing deficiencies in unplanned patient education, more and more health professionals now advocate structured, planned, formal approaches. Traditionally, patient education has been implemented by family physicians through an informal one-to-one approach or through delegation to allied health professionals. Virtually all health professionals who come in direct contact with a patient have an opportunity to provide some patient education.

Nurses provide much patient education while performing their duties. In the UKSM Department of Family Practice, a full-time nutritionist teaches patients nutritional guidelines and dietary requirements. Physical therapists teach therapeutic exercise and the use of prosthetic devices. A registered pharmacist explains purposes and possible side effects of medications prescribed. This is particularly important for patients discharged from inpatient care with prescribed medication regimens. The list of health professionals who are or could be involved in patient teaching is virtually endless.

However, the physician is the team leader and must assume responsibility for patient education. This does not require that the physician do most of the teaching, but rather that he/she set the stage for the teaching process with the patient and office or hospital staff. Patient educational processes afford the physician an opportunity to learn from patients, to improve communication techniques, and to identify the type of information and manner of presentation that will most effectively accommodate the needs of the patients. This can also motivate patients to seek earlier care while problems are minor and thus require less sophisticated therapy. It can lower readmission rates for chronically ill patients, reduce unnecessary utilization of health care professionals and facilities, and thereby reduce health care costs. There is a definite positive correlation between patient compliance and the efficiency and effectiveness of medical practice. However, the major emphasis might best be placed on building a partnership between the patient and health care professionals.[1] Other advantages include reduction of fear and anxiety in the patient population (thereby improving patient morale), shortened recovery time, and a decrease in malpractice incidents by inclusion of the patient on the health care team. Kozoll suggests several alternate uses of educational checklists for routine chronic illnesses, follow-up visits, and assessments of the compliance to regimens and medications as a standard medical plan in the treatment. He believes that if the process of patient education is to be delineated as a treatment practice, it must be accorded its place in the chart of the patient's treatment program.[2] Examples of the need for this approach have been reported with the statistic that when live oral polio vaccine first became available, 90 per cent of children in the United States between the ages of one and four years were given adequate protection. By 1973, only 60 per cent of children in this age category were adequately protected.[3] In a recent paper on a simultaneous comparison of a

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tertiary medical center clinic, a family practice clinic, and a private primary clinic setting, the author has shown essentially identical compliance when the techniques utilized were the same. In both private and institutional practice, patient noncompliance with medical instruction may result from a fundamental lack of understanding of the instructions provided.[4] In a 1974 study, Mazzullo, Lazagna, and Griner showed that 64 per cent of their patients did not understand written instructions accompanying certain prescribed medications.[5] Due to the high percentages of possibility for misunderstanding of common prescription instructions and doctor-patient communications, the Department of Family Practice at UKSM has set up patient education routines to facilitate compliance and improve communication. We utilize six methods: (1) one-to-one dialogue between health care professional and patient; (2) group experience; (3) mediated materials; (4) printed materials; (5) audio-visual materials; and (6) office and clinic utilization.

In discussing one-to-one dialogue, Bartlett has described patient education in terms of problem solving. Major factors that influence behavior involved in the relevant illness should be systematically discussed. Such factors include influences relating to the patient, to the environment, and to the family of the patient. All of these should be considered during a one-to-one dialogue, and instructions by the health care professional should be provided to family members surrounding the patient. Evaluation would then include assessment of the improvement in the patient knowledge, attitudes, comprehension, and adherence to the regimen.[6]

Group experience is employed in our clinic for hypertensive, obstetrical (pre-natal), diabetic, and obese patients.

The mediated materials used are made available by various organizations concerned with the illnesses in question, and these are usually presented by the health care professional who is directly involved with the patient's care. Printed materials are usually given to the patient by a member of the health team who is directly involved with the instruction of the patient and the materials in question. Audio-visual materials are made available to be used either on a one-to-one basis with the patient or in a group experience.

Seven areas of the practice area are utilized in the educational program: (1) the waiting room; (2) receptionist's desk; (3) lavatory; (4) examination rooms; (5) nurses' station; (6) laboratory; and (7) patient education room.

In the waiting room, printed material is available to the patient from the receptionist's desk. This material includes information on making appointments; indications for a call to the physician; how to break an appointment; how to contact the physician after hours, on holidays, or weekends; and billing and charge procedures.

In the lavatory, instructions are available explaining how to obtain clean-catch urine and stool specimens.

The examination rooms afford excellent opportunities for patient education, either by the nurse or the physician, and the nurses' station may be utilized for follow-up evaluation and assessment of the communication between the patient and the physician.

In the laboratory, the technicians may effectively instruct patients as to testing and pre-test preparation for extended laboratory tests continuing for a period of time.

In some practices, a special room centralized in the clinic facilities can be effectively utilized, but setting aside a room for the specific purpose does not in itself insure good patient education; it must be part of a plan for teaching the patient in the context of his/her specific needs. Such a room is most useful for group educational activities.

Implementation of patient education in a practice is a process that extends throughout the entire period of time the patient spends in the clinic environment. Friedman[7] suggests the following guidelines:

1. Cover the most critical points first;
2. Deal with those items of greatest importance to the patient — this will increase patient rapport with the educational regimen;
3. Give priority to information and procedures that will expedite the patient's recovery; procedures requiring practice and proficiency should periodically be re-evaluated with the patient; and
4. Schedule consultations with appropriate health care professionals so as to maximize the utilization of personnel and equipment.

The patient education regimen must be structured to increase the patient's knowledge and skills, and re-evaluation and assessment should be continual as the patient returns to the clinic periodically. Checklists kept in the patient's chart are helpful in documenting the patient's educational progress and monitoring his ability to grasp material presented. Helpful guidelines to effective patient education include:

1. Time spent with the patient by the health care professional is directly proportional to results. This type of interaction with the patient indicates a personal interest in the individual and thus increases the willingness of the patient to follow prescribed directions.

2. Personalized standard written handouts, with the patient's name written by the health care professional and with the directions written at that time, emphasize to the patient the personal nature of the information being given.

3. Patients vary in receptability to various learning techniques; therefore a variety of strategies should be employed with the patient and family members who are brought into the health care continuum. It is important that all information be presented clearly, concisely, and understandably.

In summary, we feel that patient education is a field that needs more intensive emphasis by the medical profession. The consequent improvement in rapport between health care professionals and patients, and better utilization of facilities and personnel, will contribute to shortened recovery time, decreased morbidity, and ultimately a corresponding reduction in health care costs. Patient education is an essential component of quality, primary care, and such education is the primary responsibility of the physician. Those who incorporate such educational programs into their routine will increase efficiency in their office practice. The patient in the physician's office may be in a state of anxiety or fear, and time spent by physician or other health care professional with such a patient is interpreted as caring. Patients may be better served if they first know how much we care rather than caring how much we know.

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Vox Dox

Vox Dox Editor:

There probably are not many pitfalls into which the physician is more likely to fall than slighting the physical examination. It is true that veteran clinicians can often pretty well figure out many cases from the history. But "pretty well" will not do.

I am reminded of this by one of Doctor Goering's New Year's resolutions in the January issue. "I resolve," he writes, "to do a thorough examination, for many errors in diagnosis and medical management can be made with hasty conclusions before a thorough examination is performed."

I hate to think of the times when a diagnosis appeared obvious that I have had a patient ask, "Aren't you going to examine me?" And of other times when, after giving the patient the diagnosis, physical examination forced me to change it. Doctor Goering's perspicacity is remarkable and his candor refreshing.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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Fetal Cardiac Arrhythmias

Interpretation of Fetal Heart Irregularities

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Auscultation of a normal fetal heart beat has been recognized as a sign of fetal viability and apparent well-being for more than 150 years. The advent of more sophisticated equipment and techniques has effected more precise diagnoses of abnormalities of fetal heart rate and rhythm, increasing the recognition of cardiac arrhythmias. This review stresses the importance of identification of fetal heart irregularities during pregnancy, labor, and delivery.

Bradyarrhythmias

FETAL BRADYCARDIA is traditionally considered a sign of fetal distress, and active intervention with expeditious delivery is frequently indicated. A slow, regular fetal heart rate may be indicative of congenital heart block rather than fetal distress. It is important to distinguish between these two phenomena since fetal distress must be readily recognized and treated or fetal death will frequently ensue. Congenital heart block, although it may indicate a potential for fetal compromise, is generally not associated with fetal distress during labor or delivery. The specific circumstances of pregnancy and labor should aid in this distinction. In addition, as discussed below, more specific evaluation with fetal electrocardiography, phonocardiography, and sonography will provide additional details to enable precise diagnosis.

This review has identified 58 cases of congenital heart block, although several cases were not documented by electrocardiography *(Table I)*. Nineteen cases (33%) died of complications within several weeks of birth and an additional infant died at one year of age. Eight of these cases died with clinical cyanosis. Fourteen autopsies were performed and congenital cardiac defects were recognized in 13 of them. The most consistent defect was a ventricular septal defect present in seven cases. Histologic evidence of calcification within the conduction system and absence of Purkinje fibers were also identified. Excluding seven cases lost to follow-up, 21 of the remaining 31 patients had normal growth and development without further evidence of cardiac disease or rhythm disturbance. Seven patients continue to demonstrate heart block but without evidence of clinical compromise. Only three infants experienced decreased growth or other significant health problems related to their congenital heart block.

These patients represent a rather heterogeneous group of causes for congenital heart block. The prognosis for successful survival depends on any underlying anomalies of the heart as well as the primary or secondary disturbance of cardiac rhythm. Some of these patients were identified prior to the availability of presently accepted medical and surgical therapy; and presumably survival and outcome would have been better had this therapy been available. Certainly there is a potential for major complications and even death for these infants. This must be recognized if these infants are to receive proper management and therapy to enable optimal survival. In utero cardiac deterioration generally does not occur with congenital heart block.

It has recently been recognized that infants whose mothers have systemic lupus erythematosus and other related diseases may develop congenital heart block.[2, 3] In some instances only serologic evidence of maternal disease has been present. Presumably this phenomenon arises due to immunologic factors associated with maternal disease. This possibility should be considered in the differential diagnosis of congenital heart block. Prognosis is generally good for these infants; however endocardial fibroelastosis has been reported at autopsy of some of these infants.

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TABLE I
FETAL BRADYCARDIA AND CONGENITAL HEART BLOCK

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
1. White, 1921	8 hrs prior to birth; irregular slow rate	7 lbs 1½ oz	—	Cyanotic, mongolian features; ECG with 2:1 and 3:1 CHB 5 wks — blowing sys murmur, ECG sinus rhythm 11 wks — murmur decreased, CXR-cardiomegaly, ECG — normal 5 mos — P_2 ↑ and sys murmur continues; irregular rate 9 mos — P_2 ↑ sys murmur & diastolic murmur at LSB Suspect (a) PDA (b) intermittent CHB (c) interventricular septal defect No further F/U
2. Yater,[a] 1929	2 wks prior to delivery; heart rate of 47	—	—	Cyanotic at birth; ECG showed CHB; died in cyanotic attack at 18 days; numerous anomalies at birth
3. Witt,[b] 1934	2 mos prior to delivery, heart rate of 44	—	—	Cyanotic at birth; ECG showed CHB; CXR showed cardiomegaly; died in cyanotic attack at 75 days; autopsy showed coarc of aorta and endocardial defect
4. Dippel, 1934	40 wks; rate of 50	6 lbs 10½ oz	—	ECG showed NSR with occ bigeminal rhythm and frequent vent-extrasystoles 2nd day — cyanotic attack and dyspnea; also heart rate and collapse Died at 87 hrs — autopsy showed stenosis of isthmus of aorta, patent foramen ovale; interventricular septum defect, PDA, extensive liver congestion and necrosis
5. MaClellan, 1935	Prenatal; irreg rate with 4-8 beats then long pause	4 lbs 13 oz	—	Congested face, "prune" color, persistence of irreg rhythm; died at 6 mos; autopsy showed patient foramen ovale
6. Heubner,[c] 1938	Prenatal; rate of 80	—	—	ECG at birth showed CHB; CXR — normal F/U at 11 mos — healthy without CHB
7. Ottow,[d] 1939	24 hrs prior to birth; rate of 32	—	—	ECG at birth showed CHB; CXR — slight cardiomegaly F/U at 5 mos — CHB persists
8. Geiger, 1940	Early labor; intermittent irreg rate of 58-64	3,180 gms	—	ECG at birth showed CHB with atrial rate of 120 with vent of 60; also Rt axis deviation and flat T waves X-ray — normal F/U at 5 mos — CHB persists — pt doing well
9. Thomson,[e] 1943	7 hrs prior to birth; rate of 60	—	—	1 yr — died of Stokes-Adams seizures; autopsy — no interventricular defect
10. Thomson,[e] 1943	4½ wks prior to birth; rate of 45	—	—	4 yrs — alive and healthy, CHB persists
11. Plant, 1944	9th mo; rate of 50-60 on fetal ECG	8 lbs 2¼ oz	—	Murmur at left 5 ICS, 3 hr ECG showed CHB & marked RAD, developed cyanosis & ↑ liver; 6 day — ECG showed CHB Died at 6 days — autopsy showed enlarged heart; sections failed to identify Purkinje fibers

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
12. Hammond,[f] 1944	1 mo prior to delivery; rate of 40	—	—	F/U at 7 mos — healthy
13. Jordan, 1947	5 wks prior to delivery; rate of 40-46	5 lbs 12 oz	—	ECG at birth showed CHB with atrial rate of 124 and vent of 43; sys murmur at left 3 ICS F/U at 12 mos — CHB persists
14. Sankey, 1948	1 day prior to labor; rate of 72-108	6 lbs 7 oz	—	ECG at 6 days — no CHB or arrhythmias; 6 days — sys murmur over precordium F/U at 2 mos — healthy
15. Sankey, 1948	1 day prior to labor; rate of 44	3.8 kb	—	2 days — loud murmur over precordium; 5 days — harsh sys murmur & enlarged heart on PE; ECG showed CHB with atrial rate of 130 and vent rate of 41 F/U at 1½ mos — CHB persists — child healthy
16. Riley, 1949	1 wk prior to delivery; irreg bradycardia and questionable murmur	—	—	Birth — S, is indistinct; 5 mos — ECG showed CHB; 6 mos — loud systolic murmur left 3 ICS & CXR showed cardiomegaly No further F/U
17. Harris, 1952	38 wks; rate of 60-70	7 lbs 4 oz	—	18 hrs ECG showed CHB; CXR — normal; F/U at 8 wks; CHB persisted
18. Moss, 1956	4 mos prior to delivery; rate of 56	—	—	Sys murmur at left 4 ICS at birth; CXR — normal; 2 hrs ECG showed CHB with atrial rate of 110 and vent of 50 F/U at 2½ yrs showed persistant CHB & child appears healthy — left ventricle minimally enlarged — suspect VSD
19. Redman, 1958	35th wk; bradycardia	—	—	ECG showed CHB with atrial rate of 136 and vent of 48; died at 4 days — autopsy showed interventricular septal defect
20. Connor, 1959	38th wk; rate of 64-68	4 lbs 14½ oz	—	ECG at birth showed CHB; CXR — cardiomegaly; 6 mos — appears to be developing normally
21. McNie, 1959	1 wk prior to delivery; rate of 40	6 lbs	—	Cyanotic attacks, machinery murmur over precordium; ECG at 2 hrs — showed CHB with RAD; died at 60 hours — autopsy showed enlarged heart, PDA, ASD
22. Smith, 1960	38th wk; intermittent bradycardia of 80-84; fetal ECG — CHB	8 lbs 1½ oz	—	11 hr ECG showed sinus arrhythmia with occ PVC; 2 days ECG showed NSR F/U at 2 yr — normal
23. Eyton-Jones, 1960	38 wks; rate of 40	5 lbs 12 oz	—	ECG showed CHB; CXR — cardiomegaly; died at 6 wks; no autopsy
24. Eyton-Jones, 1960	36 wks; rate of 88	7 lbs 10 oz	—	Cyanotic; ECG showed CHB with multifocal ventricular systoles at rate of 48. Died at 38 hrs; autopsy showed hypertrophy of both ventricles, septum defect; partial fusion of mitral and tricuspid values, as seen with A-V communis
25. Larks, 1960	Fetal ECG with CHB & atrial rate of 162 and vent of 51	—	—	ECG at birth showed CHB F/U — healthy
26. Larks, 1960	Fetal ECG with CHB & vent rate of 36	—	—	ECG at birth showed CHB; died at 1 wk; no autopsy
27. Larks, 1960	Fetal ECG with bradycardia	—	—	ECG at birth showed CHB; died — autopsy showed calcifications in conduction system

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
28. Dunn, 1960	30 wks, bradycardia of 52; 1 wk prior to delivery; sys murmur Fetal ECG — CHB	4 lbs 12 oz	—	Cyanosis at birth; prolonged systolic murmur; ECG showed CHB F/U shows ↓ growth with episodes of cyanosis
29. Hon, 1962	Fetal ECG shows rate of 35-70 with missed beats — sinus arrest and nodal escape	—	0	Prolapsed cord —
30. Hon, 1962	Fetal ECG with rate of 100 and sinus pauses	—	8-10	ECG — fewer pauses and few PVCs; 5 days — normal
31. Hon, 1962	Fetal ECG with rate of 60 — sinus arrest and nodal escape (one episode)	—	8-9	Normal
32. Hon, 1962	Fetal ECG with rate of 50 — sinus arrest with nodal contractions (one episode)	—	10	—
33. Gochberg, 1964	Labor, bradycardia of 27-30	6 lbs 7 oz	—	ECG at birth shows CHB; CXR shows cardiomegaly; sys murmur at left 3 ICS Died at 3½ mos after cyanotic attack — no autopsy
34. Gochberg, 1964	2 days prior to delivery; rate of 50-60	6 lbs 11 oz	—	ECG showed CHB with vent rate of 50; CXR showed cardiomegaly; II/VI sys murmur 7 yrs — CHB with incomplete RBBB and III/VI sys murmur; lost to F/U
35. Gochberg, 1964	32nd wk; bradycardia of 70-80	7 lbs 2 oz	—	ECG showed CHB with vent rate of 66; CXR — cardiomegaly, II/VI sys murmur; LSB No F/U
36. Sundaram, ?	32 wks; bradycardia of 80-82	5½ lbs	—	ECG on 3rd day showed CHB with ventricular rate of 60 No F/U
37. Kendall, 1967	38 wks; sinus bradycardia; pre-eclampsia	3,400 gm	10	Normal
38. Kendall, 1967	22 wks; bradycardia of 110; twins, polyhydramnios; diabetes, absence of heart sounds during labor	455 gm and 370 gm	5	Died at 12 hrs; secondary to immaturity
39. Kendall, 1967	37 wks; bradycardia of 108; pre-eclampsia	3,400 gm	10	Normal
40. Kendall, 1967	34 wks; rate of 106	3,120 gm	10	Normal
41. Kendall, 1967	32 wks; rate of 106	2,440 gm	10	Normal
42. Kendall, 1967	27 wks; rate of 102	3,215 gm	10	Normal
43. Kendall, 1967	35 wks; CHB with rate of 100 on fetal ECG	3,685 gm	9	Normal
44. Kendall, 1967	38 wks; bradycardia of 100	2,610 gm	10	Normal
45. Kendall, 1967	34 wks; rate of 94, polyhydramnios	2,780 gm	2	Murmur at birth; died at 7 days — autopsy showed interventricular septal defect
46. Kendall, 1967	33 wks; rate of 90	2,525 gm	10	Normal
47. Kendall, 1967	39 wks; rate of 80 with bigeminy	2,860 gm	10	Normal
48. Kendall, 1967	35 wks; rate of 75 with CHB on fetal ECG; polyhydramnios	2,240 gm	2	Died at 24 hrs; autopsy showed pulmonary hypoplasia & hypoplastic chondrodystrophy

Author, Date	*Time of Detection Abnormalities*	*Birth Wt*	*Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
49. Kendall, 1967	40 wks; rate of 66 with CHB on fetal ECG	3,090 gm	8	Normal
50. Kendall, 1967	44 wks; rate of 65 with CHB on fetal ECG	2,300 gm	1	Died at 4 hrs of respiratory distress; autopsy showed pulmonary atelectasis with subarachnoid hemorrhage
51. Kendall, 1967	36 wks; rate of 60	2,380 gm	9	Normal
52. Kendall, 1967	39 wks; rate of 56	3,580 gm	9	Normal
53. Teteris, 1968	32 wks; rate of 60; fetal ECG showed CHB	3,115 gm	8	4 yrs — healthy and Grade I/VI sys murmur at LSB
54. Sokol, 1974	After induction, difficult detection of fetal heart rate; fetal ECG showed CHB with ventricular rate of 50	—	1/5	ECG — CHB with RAD; ↑ S_1 on PE; CXR — cardiomegaly; treated with bicarbonate digoxin and isoproterenol Died at 28 hrs — autopsy showed biventricular hypertrophy, A-V communis; moderate endocardial fibroelastosis, septum secundum defect; conduction system interrupted with fibrosis in atria and at A-V level
55. Abdulla, 1975	34th wk; rate of 56 Fetal ECG — CHB	2,040 gm	—	Died at 15 min; autopsy showed multiple anomalies including dextrocardia with enlargement and VSD; situs inversus
56. Hamilton, 1977	32 wks; rate of 50 with bizarre QRS complexes; sys diamond shaped murmur; amniocentesis — L/S ratio of 1.0, estriols & sonography — normal	3,930 gm	8/8	Cyanotic at birth; 2 hr ECG showed CHB with vent rate of 55; CXR — cardiomegaly; 1-day catheterization showed common atrium, complete A-V canal, double outlet right vent and valvular and infundibular pulmonic stenosis; aneurysmal right aortic arch and interrupted left sided inf vena cava with azygous continuation Died at 9 days
57. Berube, 1978	Prior to C-section at 32 wks; rate of 66 Mother with SLE	1,780 gm	4/8	ECG showed CHB with ventricular rate of 64 6 mos — CHB persists; evidence of SLE
58. Berube, 1978	4 wks prior to delivery; bradycardia. Fetal ECG-CHB with vent rate of 60; mother with SLE	3,220 gm	8/8	ECG at birth — CHB; CXR and echocardiography — normal 15 mos — normal with CHB; no evidence of SLE

Tachyarrhythmias

Fetal tachycardia may also be a sign of fetal distress. Maternal hyperpyrexia and medications may also produce fetal tachycardia and their influence must be identified. Tachycardia may also arise from disturbances of fetal cardiac rhythm without evidence of fetal distress. Fetal electrocardiography will aid in this distinction.

Forty-six cases of fetal tachycardia secondary to supraventricular foci and atrial flutter have been identified *(Table II)*. Several cases were not documented by electrocardiography. In 28 cases (61%) signs (cardiomegaly, edema, or ascites) of congestive heart failure were present at birth. Thirty-two cases were successfully converted to a normal sinus rhythm with digitalis, quinidine, or lanatocid. One case converted with bilateral carotid message, and three cases required direct current electroshock therapy for conversion.

At follow-up, 32 cases demonstrated a normal sinus rhythm with no evidence of sequelae. Four cases were lost to follow-up. Six infants continued to exhibit evidence of disease. Two patients had recurrence of tachycardia. The remaining four patients all had structural cardiac disease (atrial septal defect — three cases, teratology of Fallot — one case). Four infants died. Two of the infants demonstrated endocardial fibroelastosis at autopsy, and a third child

had a rhabdomyoma of the heart.

Fetal tachycardias may be manifest as congestive heart failure in utero or in the immediate neonatal period. Prompt recognition of this arrhythmia and treatment with digitalis or other pharmacologic agents is imperative. Cardioversion may be necessary in the neonate resistant to medical therapy. Failure of cardioversion to return a normal sinus rhythm usually signifies underlying anatomical cardiac disease. In the absence of structural cardiac anomalies, prompt therapy is generally associated with a good prognosis.

TABLE II
FETAL TACHYCARDIA — SUPRAVENTRICULAR TACHYCARDIA AND ATRIAL FLUTTER

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
1. Carr, 1931	3 wks prior to birth; irreg rate of 160	10 lbs 13 oz —	ECG — rate of 192 with 2:1 and 3:1 flutter — lasted 10 days. CXR — cardiomegaly; cyanosis and marked restlessness; sys murmur at LSB F/U at 4 mos — normal
2. Tollas,[g] 1933	24 hrs prior to birth; irreg rate of 200	— —	No ECG; normal at 6 wks
3. Sherman,[h] 1934	8th mo; rate of 190	7 lbs 8 oz —	ECG at 2 mos — rate of 232 with 2:1 flutter; CXR — cardiomegaly; dyspnea and cyanosis at 1st and 4th mo; received digitalis at 3 mos F/U at 5 mos — normal
4. Tarnower,[i] 1942	8th mo; irreg rate of 190	8 lbs 12 oz —	ECG 1st day — rate of 195 with flutter and irreg block; CXR — normal; color poor; arrhythmia lasts 3 days — pt treated with digitalis F/U at 10 yrs — Normal
5. Hedberg, 1945	3 wks prior to delivery; irreg rate of 180	8 lbs 12 oz —	2 hr ECG showed flutter with 2:1 and 3:1 block; CXR — cardiomegaly; intermittent harsh sys murmur; treated with digitalis F/U at 2 mos — normal
6. Taussig,[j] 1947	Prenatal; rapid rate	— —	Flutter with block on ECG; successfully treated with digitalis; F/U at 5 yrs — evidence of interatrial septal defect
7. Garvin, 1947	6th mo; rate of 200-250	8 lbs 1 oz —	2nd day ECG — AV nodal tachycardia CXR — cardiomegaly; treated with digitalis and quinidine; arrhythmia intermittent for 3½ yrs F/U at 5 yrs — normal
8. Stadler,[k] 1948	8th mo; irreg rate of 136-208	7 lbs 13 oz —	ECG on 6th day — flutter with 2:1 block; cardiomegaly; sys murmur; treated with quinidine and digitalis F/U at 9 mos — normal
9. McClean,[l] 1952	Prenatal; rapid irreg rate	8 lbs 14 oz —	Slight cyanosis; 3rd day ECG — rate of 200 with flutter and irreg block; enlarged heart — 1 mo; treated with digitalis and quinidine F/U at 1 yr — normal
10. Sancetta, 1952	Labor, rate of 240	7lbs 11 oz —	1 hr ECG — SVT of 222; treated with bilateral carotid massage which terminated arrhythmia F/U at 9 mos — normal

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
11. Anderson, 1953	3 wks prior to birth; irreg rate rhythm	7 lbs 4 oz	—	2 hr ECG — rate of 190 with flutter and irreg block; dyspnea and cyanosis; cardiomegaly; sys murmur over base; occ premature beats; treated with digitalis — arrhythmia lasted 4 days F/U at 1 yr — normal
12. Mack, 1954	Labor; regular rate of 240	7 lbs 14 oz	—	Cyanosis; resp distress; treated with digitalis; NSR at 3½ days with paroxysmal tachycardia 6 wks old — tachycardic episode with return to NSR after digitalis 3½ mos — tachycardia with return to NSR F/U at 15 mos — normal
13. Hilrich, 1955	30 wks; irreg rate of 200	8 lbs 4 oz	—	Cyanosis, dusky skin — ECG showed nodal tachycardia of 300; CXR — normal; treated with digitalis; 1 wk — NSR 3 mo ECG — slight sinus tachycardia
14. Segal,[m] 1956	3 days prior to delivery; rate of 200	9 lbs 1 oz	—	Cyanosis of extremities; CXR at 10 hrs — cardiomegaly; treated with digitalis F/U normal
15. Siderides, 1957	6th mo; irreg rapid rate	8 lbs	—	Cyanosis; ECG with flutter and 4:1 block; CXR — cardiomegaly; grade II/VI sys murmur; treated with digitalis Died at 2½ mos — autopsy showed subendocardial fibroelastosis of both ventricles and auricles; ectopic skeletal muscle in S-A node; foramen ovale closed
16. Kesson,[n] 1958	Labor; rate of 228	9 lbs 1 oz	—	Edema and ascites; treated with digitalis F/U — normal
17. Hassenrück,[o] 1965	2 mos prior to delivery; rate of 180	6 lbs 8 oz	—	Peripheral cyanosis; defibrillated → NSR F/U — normal
18. Kendall, 1967	32 wks; tachycardia of 200	1,935 gm	10	Normal
19. Kendall, 1967	36 wks; tachycardia of 200	3,375 gm	10	Normal
20. McDonagh, 1968	Labor; irreg rate of 200	3,800 gm	10	1 day ECG — atrial flutter with rate of 420 and 2:1 block; CXR — cardiomegaly; treated with digitalis and converted at 6 days F/U at 10 wks — normal
21. McDonagh, 1968	Labor; irreg rate of 120-160	4,110 gm	5/10	2 day ECG — flutter with 2:1 block; 5 day — sys murmur; 8 day ECG — variable nodal rhythm; treated with digitalis F/U at 7 wks — normal
22. Blumenthal, 1968	Labor; fetal ECG shows irreg rate at 420 with block from 2:1-4:1	9 lbs 9 oz	9	21 hr ECG showed flutter at 2:1 block; CXR — normal; treated with digoxin F/U at 48 hrs — normal
23. Silber, 1969	4½ wks prior to delivery; rate of 240-320	3,370 gm	2/5	ECG showed NSR; massive edema at birth; 5th day ECG—atrial tachy 240 5th-10th day — atrial tachy and flutter with intermittent block; treated with digitalis and quinidine F/U at 3 mos — normal
24. Levkoff, 1969	Labor; rate of 250	6 lbs 6 oz	—	Cyanotic and tachypneic; ECG at 3 hrs — SVT with rate of 260; treated with digitalis; 7 hrs — NSR F/U at 10 wks — normal

Author, Date	*Time of Detection Abnormalities*	*Birth Wt*	*/Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
25. Levkoff, 1969	3 wks prior to delivery; irreg rate of 120-240	7 lbs 8 oz	—	3 day ECG showed atrial flutter with 2:1 block; treated with digitalis F/U at 7 yrs — normal
26. Tuxen, 1971	Labor; intermittent rate of 200-280	4,025 gm	7/9	EKG and CXR at birth — normal F/U at 6 mos — normal — questionable LVH
27. Barclay,[p] 1972	Prenatal; tachycardia	3,800 gm	—	EKG — atrial flutter with variable block; ASD with mild coarctation of the aorta
28. Hurwitz,[q] 1973	Tachycardia; prenatal	—	—	EKG — atrial flutter ASD
29. Herin, 1973	Prenatal; rate of 160-200	—	8/10	6 hr ECG — paroxysmal SVT with 2:1 block and rate of 160 to 200; heart failure present — treated with lanatocid F/U at 10 mos — normal
30. Herin, 1973	Prenatal; rate of 140	—	5/5	5 hr ECG — paroxysmal SVT with 2:1 block and rate of 190; ventricular fibrillation; questionable WPW syndrome; heart failure present — treated with lanatocid Died at 9 hrs — autopsy showed enlarged heart
31. Herin, 1973	Prenatal; rate of 140-200	—	4/10	1 hr ECG showed flutter with 2:1, 3:1, and 4:1 block; heart failure present; treated with digitalis F/U at 20 mos — normal
32. Herin, 1973	Prenatal; rate of 140-175	—	10/10	6 mo ECG showed 2:1 and 3:1 block with atrial flutter; ventricular fibrillation; treated with DC shock F/U at 12 mos — normal
33. Herin, 1973	Prenatal; rate of 180	—	4/10	12 hr ECG — paroxysmal SVT at rate of 210; treated with digitalis F/U at 18 mos — normal
34. Herin, 1973	Prenatal; rate of 100-180	—	10/10	1 hr ECG — paroxysmal SVT with variable block and rate of 120 F/U at 6 mos — normal
35. Herin, 1973	Prenatal; rate of 200-250	—	9/10	6 hr ECG — paroxysmal SVT with variable block and rate of 150 to 230; heart failure present; treated with lanatocid and digitalis Died at 8 wks — autopsy showed enlarged heart with idiopathic pulmonary hypertension and right heart failure; also atrial fibroelastosis
36. Herin, 1973	Prenatal; rate of 120-200	—	4/9	4 hr ECG — irreg rate of 120 to 205 with paroxysmal SVT; vent hypertrophy; heart failure present; treated with digitalis Died at 4 mos — autopsy showed enlarged heart with rhabdomyoma
37. Radford, 1976	7th mo; tachycardia	4,540 gm	—	Cardiac failure with edema and cyanosis at birth — treated with digitalis and diuretics No F/U
38. Radford, 1976	Prenatal; irreg tachycardia	2,950 gm	—	Heart failure with cyanosis at birth — ECG showed SVT with rate of 220; treated with digitalis No F/U

Author, Date	*Time of Detection Abnormalities*	*Birth Wt*	*Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
39. Radford, 1976	Prenatal; irreg tachycardia	2,980 gm	—	ECG showed atrial flutter with 2:1 block at rate of 210; hypoglycemia; treated with glucose and digitalis No F/U
40. Radford, 1976	Prenatal; irreg tachycardia	—	—	Heart failure; cyanosis; ECG-SVT; hypoglycemia; treated with glucose, digitalis and cardioversion; catheterization showed normal heart No F/U
41. Radford, 1976	Prenatal; irreg tachycardia	4,200 gm	—	Ascites, heart failure; ECG-SVT at 300; treated with cardioversion, digitalis, diuretics, propranolol and diphenylhydantoin Catheterization showed — VSD and poor myocardial function SVT — recurred
42. Radford, 1976	Prenatal; irreg tachycardia	3,100 gm	—	Cyanosis, edema, heart failure, hypoglycemia; ECG showed atrial flutter with rate of 200; treated with digitalis, diuretics, and cardioversion No recurrences
43. Hochberg, 1976	Labor; rate of 150-300; fetal ECG — paroxysmal SVT	—	—	ECG — normal F/U — normal
44. Pearl, 1977	3 wks prior to delivery	3,700 gm	—	Cyanotic, massive edema, III/VI holosystolic murmur; ECG — atrial flutter with variable block; CXR — cardiomegaly; treated with digitalis Catheterization showed tetralogy of Fallot
45. Hughey, 1977	Labor; rate of 320	2,663 gm	9/10	15 min ECG-SVT at 240 4 hrs — normal
46. Sugarman, 1978	36 wks; rate of 180; fetal ECG-SVT; acid/base balance — normal	2,630 gm	9/9	ECG — rate of 180-220; congestive heart failure present Tachycardia remained for 6 wks

Extrasystole

Premature cardiac contractions may be of either atrial or ventricular origin. As in many adults, occasional premature contractions are not generally associated with the presence of disease. In most instances, extrasystoles are supraventricular and not ventricular in origin. The precise cause for the occurrence of such irregularities in utero is not certain; however Hon and Huang have suggested a vagal disturbance as important.[1]

Fifty-six cases of premature beats with irregular rhythms have been reported in the literature *(Table III)*. Excluding one case lost to follow-up, 50 cases had no evidence of compromise at follow-up examination. Two infants continued to exhibit paroxysms of tachycardia with continued blocked atrial systoles. One of these infants had aortic anomalies. Three infants died, and all exhibited multiple cardiac anomalies at autopsy. In the absence of structural cardiac anomalies, prognosis is excellent for these patients. Ninety per cent of all infants survived, and the cardiac rhythm disturbance generally spontaneously subsided.

TABLE III
PREMATURE ATRIAL AND VENTRICULAR CONTRACTIONS AND BLOCKED CONTRACTIONS

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
1. Dippel, 1934	39 wks; frequent extrasystoles; loud sys murmur	5 lbs 13¼ oz	—	12 hrs — cyanosis, edema — sys in at left 3 ICS and left 5 ICS Died at 60 hrs Autopsy showed transposition of great vessels; patent foramen ovale, rudimentary tricuspid valve; small patent interventricular septum; small hemangiomas on mitral valve; small left ventricle; hypertrophied right ventricle; PDA; marked pulmonary artery stenosis
2. Roberts, 1938	3 wks prior to delivery; irreg rhythm with freq extrasystoles and pause; soft sys murmur; mother with congenital stenosis of aortic arch	—	—	Cyanotic, murmur, and arrhythmia continue as before birth Died at 42 hrs — autopsy showed patent interatrial septum and bicuspid aortic valve
3. Fenichel, 1942	8th mo; irreg series of 3 beats followed by pause	—	—	Runs of 2-4 beats with pause; ECG days — RAD with blocked left atrial extrasystoles 2 mos — harsh sys murmur, ↑ over pulmonic area 9 mos — CXR — left heart enlarged with widened aorta and ↑ vasc markings No further F/U
4. Redman, 1958	Labor; rate of 80	—	—	ECG — extrasystoles at 3/min; 2 mos — occas extrasystoles
5. Redman, 1958	Labor, irregular rate	—	—	Freq atrial premature beats F/U at 1 yr — normal
6. Redman, 1958	Labor; premature beats	—	—	Premature beats continue F/U at 1 mo — normal
7. Redman, 1958	Labor; premature beats	—	—	Normal
8. Redman, 1958	Labor; premature beats	—	—	Premature beats continue F/U at 4 days — normal
9. Redman, 1958	Labor; dropping every 4th beat	—	—	ECG; nodular atrial extrasystoles F/U at 10 mos — normal
10. Redman, 1958	Labor; premature beats	—	—	Premature beats continue F/U at 3 mos — normal
11. Redman, 1958	Labor; rate of 60-112	—	—	ECG — atrial extrasystoles 1 in 3; 4 mos — extrasystoles continue F/U at 4 yrs — normal
12. Freistadt, 1962	Labor; rate of 120-144; fetal ECG — bigeminal rhythm	—	7	Normal
13. Hon, 1962	Labor; rate of 150 with premature contractions; lasts 5 min	—	10	Normal
14. Hon, 1962	Labor; rate of 150 with supraventricular premature contractions at 5/min	—	8/10	Normal
15. Hon, 1962	Labor; rate of 140 with supraventricular PC at 10/min	—	10	Normal
16. Hon, 1962	Labor; rate of 130 with ventricular PC at every other beat	—	9	1st day PVC F/U 2nd day — normal

Author, Date	*Time of Detection Abnormalities*	*Birth Wt*	*Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
17. Hon, 1962	Labor; rate of 80 with supraventricular PC at 1-5/min	—	7/10	Normal at 7 days
18. Hon, 1962	Labor; rate of 120 with supraventricular PC at 10-48/min	—	8/10	Normal at 20 min
19. Hon, 1962	Labor; rate of 160 with supraventricular PC at 4/min	—	6/8	Normal
20. Hon, 1962	Labor; rate of 115 with SVPC at 2/min; also sinus pause	—	—	5 hr — occas PC F/U at 5 days — normal
21. Hon, 1962	Labor; rate of 105 with SVPC every other beat; also sinus pause	—	9/10	Normal at 37 min
22. Hon, 1962	Labor; rate of 130 with supraventricular PC; lasts 25 min	—	9/10	Normal
23. Hon, 1962	Labor; rate of 110 with SVPC; lasts 4 min	—	9/10	Normal
24. Hon, 1962	Labor; rate of 150 with SVPC at 3/min; also sinus pause	—	7/10	SVPC 1st few min
25. Hon, 1962	Labor; rate of 140 with SVPC; lasts 15 sec	—	9/10	Normal
26. Hon, 1962	Labor; rate of 125 with SVPC of 9/min	—	8/10	Normal at 2 days
27. Hon, 1962	Labor; rate of 60 with SVPC; lasts 24 sec	—	9/10	Normal
28. Hon, 1962	Labor; rate of 130 with SVPC	—	9/10	Normal
29. Hon, 1962	Labor; rate of 120 with SVPC; lasts 30 min	—	7/9	Normal at 11 hrs
30. Hon, 1962	Labor; rate of 140 with SVPC; lasts 20 min	—	1/6	No F/U
31. Hon, 1962	Labor; rate of 140 with SVPC at 10/min	—	10	Normal
32. Hon, 1962	Labor; rate of 130 with vent PC every 2-3 beats	—	8/10	Infrequent PVC Normal at 48 hrs
33. Hon, 1962	Labor; rate of 130 with SVPC; lasts 20 min	—	10	Normal
34. Kendall, 1967	24 wks; fetal ECG shows PAC	2,100 gm	—	F/U at 3 yrs — healthy
35. Komáromy, 1967	Prenatal; premature beats	—	8	Normal
36. Komáromy, 1967	Delivery; premature beats	—	10	Normal
37. Komáromy, 1967	Delivery; SVPC	—	10	Normal
38. Komáromy, 1967	Delivery; PVC; acid-base normal	3,500 gm	9	PVC for 24 hrs
39. Komáromy, 1967	Delivery; SVPC, bigeminy; trigeminy	3,050 gm	10	Premature beats with SA block for 21 days F/U at 4 mos — normal
40. Bernstine, 1968	Delivery; rate of 200 with episodes of bigeminy	4 lbs 2½ oz	—	Normal rate in 3 days F/U at 6 mos — normal

Author, Date	*Time of Detection Abnormalities*	*Birth Wt/Apgar*		*Symptoms, Treatment, Follow-up, and Findings*
41. Iyengar, 1972	30th wk; rate of 100 with frequent PC; SEM; phonocardiography — S_1nl; S_2 single fetal ECG — bradycardia and frequent PC followed by pause	4 lbs 14 oz	—	Frequent PC; systolic thrill left 2 ICS and 3 ICS; S_1nl; S_2 single; sys murmur over precordium ECG — bradycardia — right atrial and right ventricular enlargement; CXR — cardiomegaly Died — 7th day; autopsy showed hypoplastic left heart syndrome; hypoplastic ascending aorta; hypoplastic left coronary artery; myxomatous charges of pulmonic valve with mild stenosis; post stenotic-dilatation of pulmonary artery; PDA; right atrial enlargement and right ventricular hypertrophy; redundant septum primum
42. Klapholz, 1974	Labor; irreg rate of 80-128; fetal ECG-PC with bursts of SVT	—	8/9	ECG — SVPC; frequent paroxysms of tachycardia at 7 wks
43. Eibschitz, 1975	Labor; irreg rate; fetal ECG — PVC as bigeminy, trigeminy, and quadrigeminy	—	—	ECG at birth — paroxysmal tachycardia then NSR; mother given sedatives, propranolol during delivery without change in fetal ECG; also given propranolol which converted fetal ECG to NSR F/U at 1 yr — normal
44. Abinader, 1976	Labor; rate of 65-70; fetal ECG shows blocked PAC with some episodes of conducted PAC → bigeminy; Acid/base — normal	—	—	ECG — atrial bigeminy; 1 wk — NSR F/U — normal
45. Abinader, 1976	Labor; fetal bradycardia; fetal and CG shows PVC with bigeminy; acid/base — normal	—	—	ECG at birth — normal; treated with propranolol to mother → NSR in fetus F/U at 6 mos — normal
46. Abinader, 1976	Labor; fetal ECG — PAC conducted aberrantly to ventricle, trigeminy, phonocardiogram — confirmatory; acid/base — normal	—	—	ECG at birth — normal; mother treated with pethidine phenergan → NSR in fetus F/U — normal
47. Schneider, 1977	Labor; rate irreg at 140; fetal ECG — PAC; preeclamptic	7 lbs 7 oz	8/9	30 min ECG — NSR 3 hr ECG — occasional SVPC with NSR; CXR — mild cardiomegaly 2 day ECG — ↓ voltage over left precordium; NSR with occasional SVPC 5 day — normal F/U at 8 wks — normal
48. Harrison, 1977	Labor; irreg rate of 70-130; fetal ECG — atrial bigeminy of 60-70	2,410 gm	8/9	ECG at birth — PAC occ; phonocardiography — normal F/U — normal
49. Webster, 1977	39 wks; intermittent rate of 60-80; fetal ECG blocked atrial; extrasystoles	2,275 gm	6/9	30 min ECG — atrial ectopies with trigeminy 2 day ECG — intermittent atrial arrhythmia 3 day ECG — NSR F/U at 17 days — normal
50. Dgani, 1978	Labor; rate of 68-74; fetal ECG — blocked PAC	3,260 gm	9/10	ECG — blocked PAC F/U at 1 mo — bradycardic — doing well

Author, Date	*Time of Detection Abnormalities*	*Birth Wt*	*Apgar*	*Symptoms, Treatment, Follow-up, and Findings*
51. Sugarman, 1978	40 wks; multiple PAC mild pre-eclampsia	2,960 gm	9/10	ECG — multifocal PAC F/U at 4 days — normal
52. Sugarman, 1978	40 wks; multiple PAC severe pre-eclampsia; acid/base — normal	3,300 gm	4/9	ECG — PAC F/U at 24 hrs — normal
53. Sugarman, 1978	42 wks; fetal ECG — mult PAC with episodes of rate of 90 after para-cervical block; acid/base — normal	3,230 gm	9/9	5 days — no arrhythmia 1 mo — notched QRS 6 mo ECG — normal
54. Sugarman, 1978	38 wks; irreg rhythm; fetal ECG — episodes of bigeminy, sinus arrest and PAC; acid/base — normal	2,490 gm	8/10	ECG — PAC F/U at 72 hrs — normal
55. Sugarman, 1978	40 wks; irreg rhythm; fetal ECG — atrial and ventricular ectopic foci; acid/base — normal	4,100 gm	3/9	Arrhythmia persists — 5 days F/U — normal
56. Sugarman, 1978	35 wks; irreg rhythm; 39 wks; fetal ECG shows PAC; acid/base — normal	3,440 gm	8/9	ECG for 3½ min after birth showed SVT of 250 5 days ECG — PAC F/U — normal

Conclusions

Fetal cardiac arrhythmias identified during pregnancy and labor may be an important indication of the fetus at risk. The possibility of fetal distress requiring immediate intervention must be considered and excluded. Once this has been done a more thorough investigation of the cardiac disturbance may be undertaken. The fetal electrocardiogram may be recorded through the maternal abdomen with appropriately adapted equipment such as the Corometrics Model 131 Fetal ECG recorder.[5] The fetal phonocardiogram may be obtained and is occasionally useful as an aid in identifying structural cardiac disease.[6] A careful sonographic evaluation of the fetus with both gray scale and real time equipment is useful also to identify evidence of fetal cardiac anomalies.[7]

The specific type of rhythm disturbance identified with the fetal electrocardiogram will enable more precise management. As indicated in this review, congenital heart block generally goes not compromise the fetus in utero, but congenital cardiac anomalies are frequently present. Tachyarrhythmias, on the other hand, frequently do produce in utero congestive heart failure, and pharmacologic therapy during pregnancy or early delivery may be necessary to maximize fetal survival. When premature contractions are identified, the fetal outcome is generally excellent, although about ten per cent of such infants have structural cardiac disease.

Mothers with evidence of fetal cardiac arrhythmias should if possible deliver at a facility with appropriate cardiac and surgical expertise. The immediate availability of consultant personnel and facilities that frequently deal with the neonate with cardiac distress will enable optimal chances for neonate survival. Immediate medical and surgical therapy for these high risk neonates will provide optimal outcome. In fact early diagnosis during pregnancy may enable therapy to begin sooner and further minimize fetal cardiac strain. Such diagnostic procedures may generally be performed at facilities that provide care for high risk pregnancies. Decisions regarding therapy and site of delivery may then be made following consultation between the primary physician and consultant.

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Puzzling Liver Nodules

Important Diagnostic Aspects of Focal Nodular Hyperplasia and Liver Cell Adenoma

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FOCAL NODULAR hyperplasia (FNH) and liver cell adenoma (adenoma) have recently gained extensive medical attention because their incidences are increasing in association with oral contraceptive use.[1-6] However, their classification in the literature is very confused. This results from poor understanding and numerous appellations for these lesions (*liver cell adenoma:* benign hepatoma, hepatoma, solitary adenoma, and hepatoadenoma; *focal nodular hyperplasia:* adenoma, benign hepatoma, focal cirrhosis, hamartomatous cholangiohepatoma, hepatoma, isolated nodules of regenerative hyperplasia, mixed adenoma, solitary hyperplastic nodule, and hepatic adenoma).[7-9] This has also resulted in confusion regarding the clinical management of patients with these lesions.[10-12]

We recently saw two patients with FNH who were prospectively evaluated with ^{99m}Tc sulfur colloid liver scans and angiography. The purpose of this paper is to report our experience with these cases and to discuss the important clinical aspects in the management of young women with suspected liver tumor.

Case Reports

Case One: A 48-year-old white female was noted to have a mass in the right lobe of the liver by surgical palpation at the time of total abdominal hysterectomy for uterine fibroids. She had experienced vague right upper abdominal discomfort for approximately nine months prior to admission. She had been taking 1.25 mg of Premarin daily for the previous four years. She denied a history of weight loss, hepatitis, or jaundice.

Physical examination revealed no palpable abdominal mass nor other abnormality. Routine laboratory studies yielded results that were within normal limits. Results of liver function tests were also within normal limits except for a slightly depressed A/G ratio.

A ^{99m}Tc sulfur colloid liver-spleen scan showed homogeneous uptake of the radionuclide in the liver and spleen *(Figure 1A)*. A selective superior mesenteric arteriogram demonstrated a 5 x 5 cm hypervascular tumor in the right lobe of the liver *(Figure 1B)*. The tumor showed a dense blush but no arteriovenous shunting. The arteriographic diagnosis was adenoma or focal nodular hyperplasia.

Focal nodular hyperplasia and liver cell adenoma are histologically different, but are similar in incidence, causes, clinical presentation, and treatment. The detection of a necrotic or hemorrhagic component in either lesion is important for the prevention of catastrophic hemorrhage or the avoidance of unnecessary liver surgery, particularly in young oral contraceptive users. Appropriate radiological evaluation of these patients is discussed.

Two mCi of ^{99m}Tc sulfur colloid was administered intravenously just prior to exploratory laparotomy. At surgery a 5 cm mass was found in the right lobe of the liver and was reported as an adenoma by frozen biopsy, and a right lobectomy was performed. The excised right hepatic lobe disclosed a circumscribed, non-encapsulated tumor measuring 4.5 x 4.0 cm. No necrosis or hemorrhage was present in the tumor. Multiple samples of the tumor and the normal parenchyma were taken prior to fixation and assayed for radioactivity. The data showed approximately 70 per cent uptake of radionuclide by the tumor/gm of tissue as compared to surrounding normal parenchyma.

The microscopic findings included a central area of scarring with irregular radiating septa and multiple hepatocellular nodules with multinucleated giant cells, fatty metamorphosis of liver cells, and disorganized proliferating bile ducts *(Figure 1C)*. The final diagnosis was focal nodular hyperplasia. The

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Figure 1A



Figure 1B

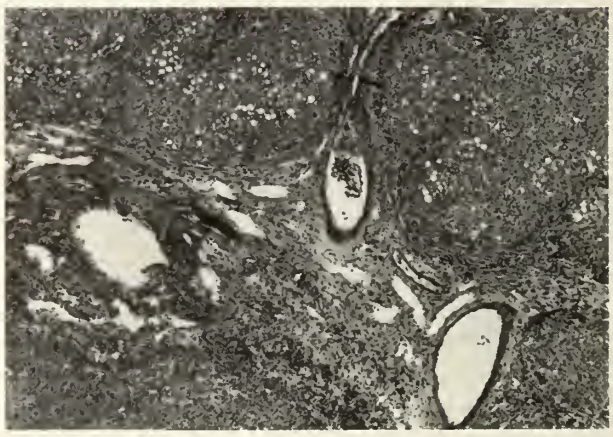


Figure 1C

Figure 1. Case One. (A) Anterior view of ^{99m}TC sulfur colloid liver-spleen scan revealing no evidence of focal defects. (B) Hepatic arteriogram demonstrating a large vascular tumor in the right lobe (arrows). (C) Focal nodular hyperplasia demonstrating central area of scarring with irregular radiating septa and multiple hepatocellular nodules (H.E. stain, ×350).

postoperative course was stormy and the patient died on the fifth postoperative day.

Case Two: A 36-year-old healthy female with an eight-year history of oral contraceptive use (Ortho Novum) was found to have a 3 cm nodule in the right lobe of the liver during surgery for tubal ligation. A ^{99m}Tc sulfur colloid liver scan was normal *(Figure 2A)*. She was advised to discontinue the oral contraceptive by her physician. Approximately eight months later during a total abdominal hysterectomy, the liver nodule was again palpated and measured 5-6 cm in size. A biopsy was performed and the diagnosis was focal nodular hyperplasia. The followup liver scan 11 months after the initial scan was again normal *(Figure 2B)*. The patient has been asymptomatic for the past two years.

Discussion

Liver cell adenoma and focal nodular hyperplasia are histologically two different benign tumors of the liver.[7] The causes, clinical presentations, and results of treatment of these lesions have been poorly understood because the lesions were rare prior to 1960.[4-7] However, with increasing incidence of these lesions reported in women using oral contraceptives, their clinical and pathological characteristics have begun to be understood.[1-3, 13]

A literature review identified several similarities between these two lesions. Exogenous hormones — particularly oral contraceptives — can induce either adenoma or FNH.[1-6, 8-13] Either lesion can present in one of three ways: (1) a mass is discovered accidently by the patient or the physician during routine physical examination or at surgery for another indication (Cases 1 and 2); (2) varying degrees of upper



Figure 2A



Figure 2B

Figure 2. Case Two. (A) Initial scan showing a uniform uptake of ^{99m}Tc sulfur colloid. (B) Normal followup scan 11 months later.

abdominal pain or discomfort is experienced with or without anorexia, nausea, or vomiting; or (3) a sudden life-threatening intra-abdominal hemorrhage occurs.[1, 3, 5, 8]

The majority of cases, whether FNH or adenoma, have been treated by resection of the tumor or partial hepatectomy with discontinuation of oral contraceptive use.[1, 5, 13] In many of those cases, one or more lesions were left behind in the remaining liver.[8, 10, 14-16] Most of these cases remained asymptomatic. Recurrence of intraperitoneal hemorrhage was not noted in any case. Regression or complete disappearance of the tumors has been reported.[14, 16, 17] Therefore, the most important clinical responsibility in evaluating a young woman with suspected liver tumor is to detect a tumor or tumors (often multiple), to determine their benign nature, and the potential for possible rupture and hemorrhage secondary to necrosis.

The radiocolloid liver scan has played an important role in the evaluation of these patients.[15, 19] Since the majority of mass lesions of the liver including adenoma are deficient in reticuloendothelial cells, the scans are usually abnormal. As shown in our cases, the radiocolloid scans are commonly normal in FNH because Kupffer cells are present throughout the tumor. When necrosis or hemorrhage develops within the tumor, the scan may become abnormal.[3, 6, 8] Therefore, a focal defect on the liver scan is a good indicator that the tumor in question contains an area of necrosis or hemorrhage if FNH, or represents an adenoma with or without necrosis and hemorrhage. When the liver scan is normal, one should consider: (1) a tumor too small to detect; (2) FNH with minimal or no hemorrhage or necrosis; or (3) a pedunculated or superficially located tumor.[13, 15] Further radiological evaluation — including sonography, computed tomography, and selective arteriography of the liver — should be performed. Hepatic arteriography has proved to be the most accurate way to detect the presence, size, number, and location of tumors.[4, 15, 20] It also provides the anatomic information regarding the blood supply to the tumor which is very useful for surgery. Sonography and computed tomography are helpful for detection of a cystic or hemorrhagic component in the tumor.[12, 13]

When a tumor or tumors are detected, open biopsy is recommended to determine the type and benignancy of the tumors. Needle biopsy is absolutely contraindicated; it may result in catastrophic hemorrhage because FNH, adenoma, and hepatoma are usually very vascular. If the tumor is proved to be FNH or adenoma without an hemorrhagic component and the patient is asymptomatic, discontinuation of the oral contraceptive, close medical observation, and serial radiocolloid liver scans are recommended (Case 2).[10, 12, 14] Only the tumor that contains a necrotic or hemorrhagic component should be selectively resected.

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Dramamine in Labor

Potential Boon or a Possible Bomb?

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DRAMAMINE use in pregnancy and labor has been discussed in ten or more articles during the past 18 years. These reports, covering approximately 2500 patients studied by observational and double blind procedures, have led the majority of authors to attribute numerous beneficial effects to its use, the two most prominent being shortening of labor and tranquilization of the mother. One of the major features stressed by most authors was the apparent safety of the drug for both fetus and mother, and no cases were reported with increased fetal or maternal morbidity or mortality. However, the majority of papers were published before the advent of widespread utilization of fetal monitoring, and therefore very few patients have been studied under full electronic monitoring during administration of this drug. With electronic monitoring now available in most hospitals, studies have appeared showing effects on uterine contractions and on fetal heart rate. This paper will review past findings and present observations not previously published.

Materials and Methods

Patients in labor or undergoing oxytocin induction were the subjects of this study. It was planned that these patients, upon reaching cervical dilation of 4 cm, would have artificial rupture of membranes (if membranes were not already ruptured) and would have application of continuous internal monitoring including pressure catheter for uterine contractions and scalp electrodes for fetal heart rate. Also at this time, Dramamine, 100 mg, in normal saline, 20 ml, would be administered intravenously. This was to be infused with a Harvard infusion pump at a constant rate such that the patient received 100 mg/3½ minutes. It was decided to measure the time from 4 cm cervical dilation to delivery of the infant and use this as a guide in determining whether Dramamine had the reported effect of shortening labor and also to observe any changes that might occur under full electronic fetal monitoring.

Results

The results obtained while studying some 30 patients according to the protocol prompted discontinuance of the investigation. Approximately 25 per cent of these patients showed evidence not only of increased amplitude and frequency of contractions (as has been documented in other studies) but a few also showed signs of uterine hyperstimulation and indications of fetal compromise. *Figures 1* and *2* show the end of Dramamine infusion with subsequent hyperstimulation and fetal bradycardia.

A study was conducted utilizing full fetal monitoring to determine the suitability of Dramamine to ease trauma and pain during labor and delivery. The results indicate that physicians should be made aware of possible tragic side effects of this medication.

Discussion

Reference to the literature on the subject reveals that the effects of Dramamine in labor have been studied in more than 2500 patients. The consensus appears to be that beneficial effects — shortening labor and calming the patient — occur. The majority of authors also agree that the medication is quite safe for both mother and infant. The medication has been administered both intravenously and intramuscularly, usually 100 mg, and usually during a period of three minutes or more when given by the intravenous route. All but two of the papers reviewed reported a shortening of labor of 0.7-3.13 hours in primigravidas and 0.6-2.7 hours in multiparas.[1, 2] It was noted in one paper that the second stage of labor was shortened as well as the first.[3]

Two mechanisms of action have been postulated. One is an antihistamine reaction blocking release of histamine from trauma and preventing smooth mus-

From the University of Kansas School of Medicine, Kansas City, Kansas.

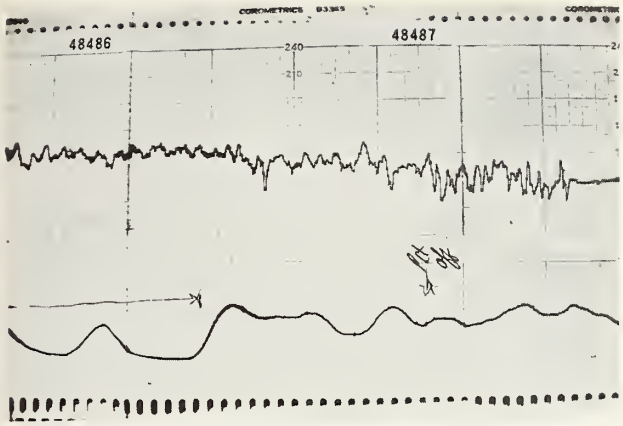


Figure 1. The end of a three minute intravenous injection of Dramamine indicated by the horizontal arrow. Following this, there is gross evidence of hyperstimulation with a dropping fetal heart rate and a loss of beat-to-beat variability. This patient was on an oxytocin infusion which was terminated at the point indicated by the vertical arrow.

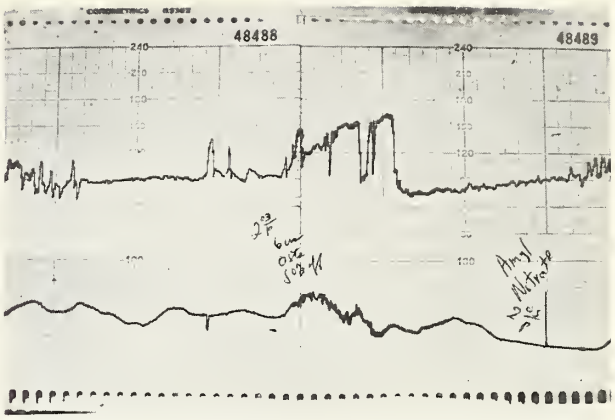


Figure 2. A continuation of the strip seen in *Figure 1*. Note that the evidence of hyperstimulation lasts some 9½ minutes. Amyl nitrate was administered just at the end of this nine-minute episode. The fetal heart rate drops as low as 75 beats/minute, and the loss of beat-to-beat variability lasts approximately six minutes.

cle spasm, thereby allowing for more rapid cervical dilation.[4, 5] The second mechanism was that of an oxytocic effect. This was noted by Watt[6] in 1961 when it was found that it was possible to use oxytocin as an induction agent for labor in dosages up to 2 mg/min intravenously. A later study showed that in vitro Dramamine indeed had an oxytocin effect on guinea pig uterine muscle as well as on human uterine muscle. It was also discovered, however, that in vitro, Dramamine acts as a relaxant on non-pregnant uterine muscles.[7] This was also seen in vivo in a number of patients who were in spontaneous premature labor and given Dramamine to stop labor. Thus, Dramamine has been advocated by some practitioners to stop false labor contractions. The theory is that Dramamine apparently has actions that vary depending on whether the patient is at term, before term, or non-pregnant.[6]

In addition to speeding of labor, another positive effect reported is that of sedation or potentiation of pain medications.[5, 8] This was noted by several authors, and again no ill effects were mentioned.

With the advent of more widespread continuous fetal monitoring, the latest papers have shown evidence of uterine hypertonus following the administration of Dramamine, but again side effects were minimized.[9] One of the older papers noted this as well.[10]

Our study began with full fetal monitoring and, as noted in the results section, approximately 25 per cent of the 30 patients experienced some evidence of uterine hypertonus. This was manifested by elevation of baseline and increase in the strength and frequency of contractions. These findings have also been noted in other published papers.[10] However, in two patients we found uterine tetany of prolonged length accompanied by profound fetal heart rate changes — including severe bradycardia — and loss of beat-to-beat variability was a much more common finding (*Figures 1* and *2*). As noted by other authors, no poor results were found when using the Apgar rating as a measure of fetal morbidity. However, in reviewing these monitoring strips, it is evident that the fetuses involved were compromised for short periods of time. Although this state of compromise was short-lived and did not recur, the long-term effects are unknown. The hypertonus was more pronounced in patients sensitive to oxytocin. One paper noted uterine tetany in pregnant guinea pig uterine muscle, but the investigators were unable to produce this in pregnant human uterine muscle.[7]

Dramamine was also given to second trimester abortion patients after they had received intra-amniotic injections of prostaglandin. These patients were monitored with extraovular balloon catheters and were noted to experience an increase in uterine contractions and pressure changes that were similar to those seen with oxytocin injection (*Figures 3* and *4*). This effect was different from that noted by other authors in that others have seen a relaxing effect on the pregnant uterus in the early stages of pregnancy. However, as our patients had also received Prostaglandin F2 Alpha, it is perhaps this substance that makes a difference in the response of uterine muscle

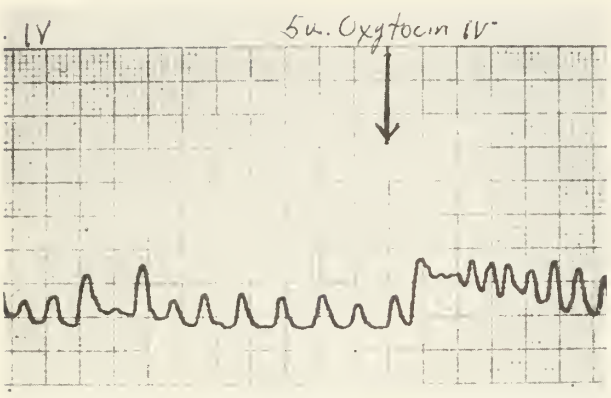


Figure 3. Tracing of a second trimester Prostin abortion patient who has already received the Prostin intra-amniotically. A 5-unit bolus of oxytocin was given intravenously, and the results are seen in a raise in the baseline in a slow return to normal during approximately five minutes.

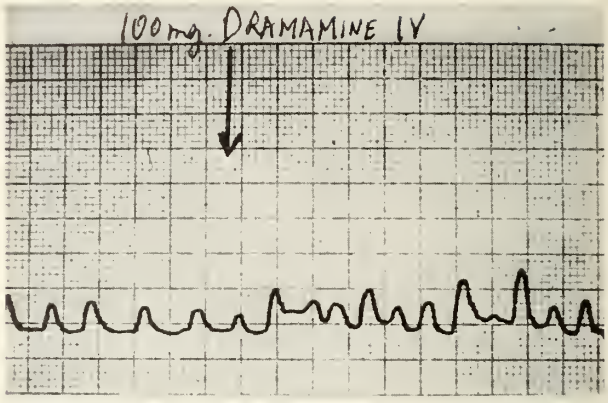


Figure 4. A 100 mg bolus of intravenous Dramamine with a subsequent rise in baseline and a return to normal during approximately three minutes. (This is also a second trimester Prostin abortion tracing.)

to Dramamine. Since Dramamine acts in a manner similar to oxytocin (as seen in these observations), we must then consider that the complications possible with oxytocin might also occur with Dramamine — that is, hyperstimulation and the possibility of uterine rupture. This then brings to mind contraindications to be considered in the use of Dramamine — such as a patient being considered for vaginal delivery after a previous cesarean section or in the patient with a compromised fetus whose placental function is already in some state of deterioration.

A beneficial effect noted in our series and not mentioned previously is that of increased tissue relaxation in the area of the perineum resulting in decreased trauma to the birth canal in those patients receiving Dramamine. This finding would tend to strengthen the initial hypothesis where blocking the release of histamine from the trauma of birth would allow for a more rapid cervical dilation by relaxation of the muscles of the birth canal.

The question is then raised as to whether pure Benadryl, because of its strong antihistamine action, would be as effective as Dramamine in its proposed results. Fifty patients were given 50 mg Benadryl intravenously during 3½ minutes. The subjective effect was similar although not as pronounced as with Dramamine, but no monitoring changes were noted. Whether this is due to the dosage of Benadryl or to the fact that this is not a combination drug is unknown at this time. Further investigation will be needed.

Dramamine is a combination of Benadryl and 8-cholorotheophylic, with Dramamine, 100 mg, being equivalent to approximately 54 mg of Benadryl.[11]

Summary

It is concluded that Dramamine has a number of beneficial effects in labor including one not previously mentioned, *i.e.* decreased trauma to the birth canal. However, it appears from these limited findings that patients must be very carefully selected, and monitoring of patients should be employed when Dramamine is administered. Because of its oxytocin-like effects, there must be constant awareness of possible adverse results that oxytocin can effect such as uterine tetany or uterine rupture. Questions raised by this investigation include the role of prostaglandin in the effect of Dramamine on labor, and whether Benadryl is perhaps as effective a medication and safer for use in labor.

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Aplastic Anemia

The Role of Bone Marrow Transplantation

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APLASTIC ANEMIA (AA) is a hematological disorder characterized by pancytopenia and markedly reduced-to-absent hematopoietic cells in the bone marrow. Although drugs, toxins, infection, and a number of clinical disorders have been associated with the marrow failure, in the majority of patients neither the cause nor the pathogenesis of the disease is understood. Based largely on the frequent successful engraftment following infusion of marrow cells from a histocompatible donor, the pathogenic role of an acquired stem cell failure has been emphasized. On the other hand, several studies have suggested a primary defect of marrow microenvironment in selected cases of marrow aplasia.

The prognosis of severe AA has been consistently poor, and the majority of patients treated conventionally die of their disease during the first year. In recent years, the introduction of bone marrow transplantation (BMT) has dramatically improved the long term survival in a large number of patients. Several studies indicate that 40-50 per cent of patients with severe AA treated with BMT regain normal marrow function and are cured of their disease.[1-3] As a result, BMT has now become the treatment of choice for severe AA if a suitable sibling donor is available. This paper summarizes the experience of our institution with allogeneic BMT in the treatment of AA and discusses briefly the major problems associated with the procedure.

Materials and Methods

Four consecutive patients with severe aplastic anemia were treated with bone marrow transplantation. Patients were considered to have severe AA if hypoplasia of the bone marrow was associated with at least two of the following: (1) granulocyte count of $<500/\mu l$; (2) platelet count of $<20,000/\mu l$; or (3) anemia with corrected reticulocyte count of <1 per cent. The pertinent clinical features and laboratory findings are shown in *Table 1*. There were three males and one female, mean age 40 years (range 22-59 yrs). Aplasia was of unknown cause in three and drug-induced in one, and the duration of aplasia ranged from six to seven months. Bone marrow transplantation was performed according to previously described methods.[4] Each recipient was prepared for transplantation with cyclophosphamide 50 mg/kg intravenously on each of four days followed 36 hours later by marrow infusion from a normal HLA-identical sibling. The median of marrow cells infused was 3×10^8 cells/kg of the recipient. All patients were given methotrexate post-grafting to modify graft-vs-host disease.

Conventional treatment of severe aplastic anemia (AA) is largely unsuccessful and most patients die within six months. Bone marrow transplantation has emerged as the treatment of choice for patients with severe AA provided a suitable donor is available. Four such cases are reviewed and the disorder and problems of treatment are discussed.

Results

All four patients survived long enough to show evidence of engraftment which occurred three to four weeks following marrow infusion. Two patients (Cases One and Two) developed severe graft-vs-host disease (GVHD) and died 66 and 61 days post transplantation. In both, high-dose prednisone and ATG failed to modify the course of the GVHD. Of the two patients, one (Case Two) developed interstitial pneumonia associated with cytomegalovirus and died of respiratory failure. One patient (Case Four) rejected his graft 134 days post-transplant and subsequently died of sepsis. One patient (Case Three) whose clinical course is depicted in *Figure 1*, is alive

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TABLE I
ALLOGENIC MARROW TRANSPLANTS FOR APLASTIC ANEMIA

Cases	*1*	*2*	*3*	*4*
Age/sex	22 yrs ♂	59 yrs ♀	25 yrs ♂	56 yrs ♂
Cause/duration (mos) of aplastic anemia	Unknown/5	Unknown/5	Unknown/7	Drug/5
Donor rel/sex	Sib/♂	Sib/♀	Sib/♂	Sib/♂
Histocompatibility	Matched	Matched	Matched	Matched
Pretransplant transfusions	RBCs — 5 units Platelets — 50 units	RBCs — 2 units Platelets — 30 units	RBCs — none Platelets — 20 units	RBCs — 4 units Platelets — 50 units
Mixed lymphocyte culture (MLC)				
Recipient → donor	>2	< .87	1	1.1
Recipient ← donor	>2	<1.76	1.23	.84
Preparation regimen	Cytoxan	Cytoxan	Cytoxan	Cytoxan
Graft take/time to engraftment	Yes/2 wks	Yes/2 wks	Yes/3 wks	Yes/3 wks
Graft-vs -host disease (GVHD)	Grade IV	Grade IV	none	none
Survival	Deceased 61 days with GVHD	Deceased 66 days with GVHD	Alive at 18 mos	Deceased 166 days with graft rejection

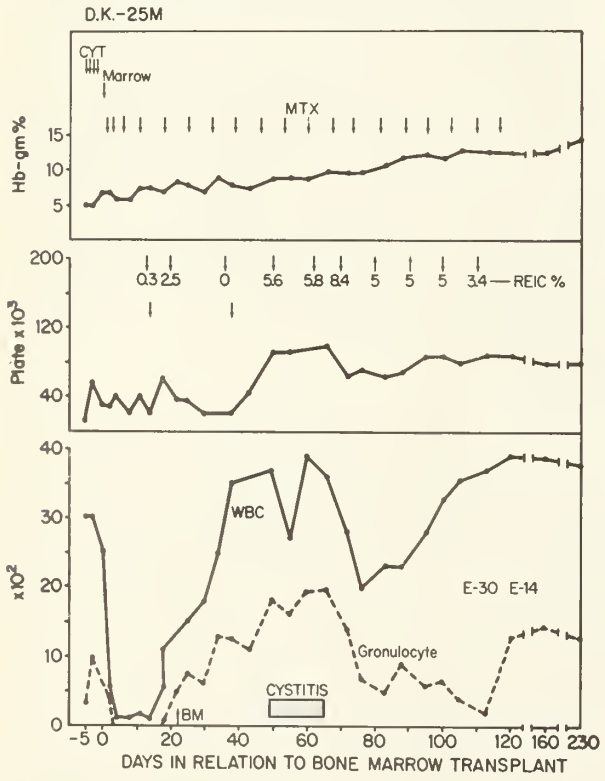


Figure 1. Days in relation to bone marrow transplant.

with complete hematological restoration 1½ years after grafting.

Discussion

Aplastic anemia is a serious disorder which frequently leads to death due to infection or bleeding. While the disease shows a variable and unpredictable course, the outcome is generally determined by the severity of the marrow failure. Thus patients with severe AA treated conventionally have an invariably grim prognosis. The majority die during the first year of their disease and very few, if any, show spontaneous recovery. In this context, a prospective randomized study has recently indicated that androgens have no beneficial effect on the course of patients with severe AA and play no role in the treatment of the disease.[1]

In recent years bone marrow transplantation has evolved as an effective form of therapy for severe AA. The largest series has been reported from Seattle where 110 consecutive patients were given bone marrow grafts from HLA identical siblings. Fifty patients regained normal marrow function and were alive 2-73 months after transplantation at the time of the report. Comparable results of 40-50 per cent long-term survival have been obtained by several centers, confirming the superiority of BMT to conventional therapy in the treatment of severe AA. Although BMT is potentially life-saving, it is a

hazardous procedure with a high rate of mortality due to the three major problems of graft rejection, GVHD, and infection.

Graft rejection occurs in approximately one-third of the patients. Despite the high incidence of this complication, it has been difficult to predict the likelihood of its occurrence before transplantation. Recent studies have, however, indicated that two factors may have important predictive value. Higher incidence of graft rejection is seemingly associated with low number of marrow cells used for transplantation ($<3 \times 10^8$ cells/kg) and the lymphocyte reactivity of recipient against donor cells as determined by relative response index (RRI) in mixed leukocyte culture. It is believed that the RRI is a measure of sensitization of recipient to donor marrow cells which may have resulted from previous exposure to blood products of the prospective donor or family members.

Graft-vs-host disease results from the immunological interaction between the immunoincompetent host and donor lymphoid cells and occurs, to a variable degree, in 60 per cent of marrow transplant patients. It involves mainly the skin, liver, and gastrointestinal tract. The lesions in the skin may vary from non-specific dermatitis to generalized erythroderma. Liver and gastrointestinal manifestations usually, but not invariably, follow the skin rash by several days. The common gastrointestinal symptoms are diarrhea and abdominal pain. Elevation of serum bilirubin, alkaline phosphatase, and transaminase are the usual manifestations of liver involvement. While the pathogenesis of GVHD is yet to be defined, the two factors that appear to correlate strongly with the presence or absence of GVHD and survival are: (1) sex match of donor and recipient; and (2) absence of refractoriness to random donor platelets at the time of transplantation.

Severe infections — due mainly to bacterial, fungal, cytomegalovirus (CMV), and pneumocystis carinii — remain major problems in aplastic patients who undergo marrow transplantation. Infectious complications are common during the period of profound leukopenia but may also occur with high frequency within the first four months of transplantation because of the compromised immune response. Interstitial pneumonitis occurs in 50 per cent of patients, and may or may not be associated with identifiable pathogens, such as CMV and pneumocystis carinii. A substantial number (50-60%) of patients with the pulmonary complication die of respiratory failure.

In our series — admittedly very small — two of the four patients died of severe GVHD. This complication occurred despite methotrexate prophylaxis and it failed to respond to high-dose prednisone and ATG. One patient rejected his graft 134 days after sustained engraftment. This patient had the strongest pre-transplant transfusion history which could have resulted in increased sensitization to donor marrow cells. One patient is well and maintains a normal hematological profile 1½ years following the transplant. In our study the long-term survival of 25 per cent (1 of 4 patients) is lower than the 40-50 per cent reported by bigger marrow transplant centers. The inferior result, however, is most likely related to patient selection. In our series, the median age of the patients was 40 years, and two were over 55 years. This is in contrast to the reported series of the various transplant centers in which the median age of the patients ranged from 4-31 years, and most of these studies did not include patients over the age of 42 years.[3] Although its role is not well defined, it is generally believed that advanced age (>45 years) may be a limiting factor in marrow transplantation. Thus it is reasonable to assume that our result was adversely affected by inclusion of the two patients over the age of 55 years.

In conclusion it is clear that BMT represents a significant advance in the treatment of AA and the physician must be cognizant of this form of therapy when a patient with AA is first seen. Thus, to explore the feasibility of BMT, HLA-typing of the patient and family should be done immediately. If a matched sibling is identified, early transplantation should be a consideration. Furthermore, to minimize the sensitization of a prospective marrow recipient, transfusions of blood products should be withheld unless absolutely indicated, and blood product support from family members should be specifically avoided. The patient should then be referred to a specialized center for therapy. If, on the other hand, a matched sibling is not available, presently marrow transplantation cannot be considered as a viable approach. Instead, conventional treatment — including synthetic androgen, corticosteroids, and long-term blood product support — should be instituted.

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Testing Their Wings

Student Perceptions of the Preceptorship Program, 1976-1979

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THE PRECEPTORSHIP was for centuries the only form of medical education, but for at least fifty years during the early part of this century, the Flexnerian influence increasingly limited student contact to the minds and hands of full-time faculty members. This, of course, cemented the foundations for the renaissance of American medicine and medical education, making America the leader in these fields. As evidenced by the collapse of diploma mills, the raising of standards, the development of evaluation systems, the growth and multiplication of specialties and the discoveries of research, the successes of the Flexnerian decades led to a partnership between federal government and what had become a successful biomedical educational establishment, eager for new challenges and new successes. America became the center for all biomedical research and education.

New problems arose from this alliance. Fein has pointed out the tensions in medical education that arose from the governmental-educational partnership and the resultant change in the mission of medical schools.[1] In 1968 the federal government began to shift its interest toward health care delivery and slightly away from health sciences research. Although preceptorships had been established as early as 1926 (Wisconsin) and also in 1951 (Kansas), there was renewed interest in them as a means of emphasizing a more people-oriented and humanistic kind of health care. There is now considerable professional and academic literature on "remote-site clinic" student experiences, largely concerned with documenting as objectively as possible what students actually learn and how that compares with the large urban teaching hospital.[2-4]

During its first twenty-four years (1951-1975), the University of Kansas preceptorship program was designed on the basis of senior medical student assignments to individual preceptors.[5-6] Beginning with the class of 1976 — through a federal grant concerned with health manpower redistribution — the program was expanded and largely regionalized,

The preceptorship is both an ancient and a modern modality for medical education. Experiences of medical students in the Kansas preceptorship program give some insights into its effectiveness as a component of the modern medical educational program.

with heightened community involvement and the development of an extensive system of evaluation. Educational objectives were established in student-preceptor workshops.[7] The program was thus redesigned to exploit the potential of a new alliance between medical school faculty, volunteer faculty, students, and communities. The hope was that each could participate, communicate, help define common goals, and benefit from the encounter. A small staff was formed to coordinate, monitor, and evaluate this encounter. On Concept Questionnaires, students were asked to give responses — both pre- and post-experience — dealing with career choice, assessment of community health resources, possible practice location, procedures done, and the preceptorship experience as a whole. Norrdin and Defoe published a preliminary study of the 1976 class.[8] Through the assistance of a research grant from the School of Medicine, all the questionnaires of some 588 students from UKSM-KC during 1976-1979 have recently been computerized, providing a group sufficiently large and varied for in-depth analysis of their experiences. Since there are some 388 variables for each of the 588 students, sophisticated analysis of the data is both possible and warranted. However, it is our purpose to present here only a descriptive

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TABLE I
RESPONSIBILITY FOR PATIENT CARE

	1976 n = 136 (%)	*1977 n = 119 (%)*	*1978 n = 159 (%)*	*1979 n = 116 (%)*
None	14 (10.3)	4 (3.4)	12 (7.5)	6 (5.2)
Limited	24 (17.6)	24 (20.2)	20 (12.6)	23 (19.8)
Moderate	33 (24.2)	37 (31.1)	67 (42.1)	47 (40.5)
Semiautonomous	64 (47.1)	53 (44.5)	56 (35.2)	39 (33.6)
Complete independence	1 (0.7)	1 (0.8)	4 (2.5)	1 (0.9)

survey of selected variables which concern the amount of student responsibility during the rotation and procedures done, student assessments of community health processes and the health care delivery system, and evaluations of the preceptorship itself including comparisons of the preceptorship with other rotations.

Student Responsibilities

During the four years, about 45-50 per cent of the students served their preceptorships in Kansas towns with less than 5,000 population, and the majority (about 70%) worked with physicians in one of the four primary care fields. Thus, the typical student served a small town, primary care rotation, though a significant minority did specialty rotations in middle-size towns and cities. Thus, clinical experiences ranged from those with a solo practitioner, to partnerships, to fairly large group clinics. In each experience, however, the emphasis was on the student's working directly with a physician on a one-to-one basis, and as a colleague rather than only as student and professor. In this context, one of the more important considerations is the amount of responsibility students have been given for patient care. These results appear in *Table I*. As can be seen, most students (71-77%) were in the "moderate responsibility" and "semi-autonomous" categories, with some indication of a trend from the latter to the former in the last two years. In any case, it is clear that students assumed a considerable degree of responsibility for patient care, which will be further indicated by responses to other questions. Most of the students who marked "none" were in specialties such as pathology and radiology which do not offer many such opportunities. Student gain in capabilities was tested by listing clinical procedures they performed, as given in *Table II*. It is evident that a number of students had considerable opportunity to learn new or unfamiliar procedures, as well as to

TABLE II
CLINICAL PROCEDURES PERFORMED BY STUDENTS DURING PRECEPTORSHIP

Procedures	*# Performed*	*%*	*# Rarely Performed Before*	*%*
	n = 588			
1. Suturing lacerations	243	(41.3)	118	(48.6)
2. First assist, general surgery	189	(32.1)	78	(41.3)
3. Deliveries	136	(23.1)	43	(31.6)
4. IVs, catheterizations, venipuncture, etc.	107	(18.2)	10	(9.3)
5. Histories and physicals	94	(15.9)	3	(3.2)
6. Sigmoidoscopy	87	(14.8)	36	(41.4)
7. Casting and setting fractures	77	(13.1)	53	(68.8)
8. Removal of warts, lesions, cancer	77	(13.1)	49	(63.6)
9. Pelvic, pap, fit diaphragm	69	(11.7)	4	(5.8)
10. Joint tap	45	(7.7)	14	(31.1)
11. Intubation	40	(6.8)	29	(72.5)
12. Circumcision	39	(6.6)	34	(87.2)
13. D & C	37	(6.3)	27	(72.9)
14. Injection	36	(6.1)	22	(61.1)
15. Others	282	(47.9)	167	(59.2)

TABLE III
STUDENT PERCEPTIONS OF MEASURES TO MAINTAIN HEALTH
n =588

	Pre	%	*Post*	%
Weight, nutrition, diet	432	(73.5)	392	(66.7)
Preventive medicine	422	(71.8)	403	(68.3)
Exercise	314	(53.4)	251	(42.7)
Smoking	211	(35.8)	179	(30.4)
Alcohol, drugs	130	(22.1)	88	(15.0)
Patient understanding	129	(21.9)	67	(11.4)
Immunization	123	(20.9)	97	(16.5)
Rest, relaxation	97	(16.5)	75	(12.8)
Compliance, follow-up	90	(15.3)	198	(33.7)
Public environment, sanitation	58	(9.9)	39	(6.6)
Hygiene	57	(9.7)	41	(7.0)
Safety	55	(9.4)	48	(8.2)
Facilities, M.D.	38	(6.5)	40	(6.8)

experience a wide variety of procedures with which they were familiar. Fundamental medical skills such as suturing lacerations and delivery of babies were further developed during this rotation.

Community Health

Several questions concerned community health and the health care delivery system. Indeed, the preceptorship appears to be the major single period in medical school in which students have the opportunity to address these matters. For example, one question asked was, "What measures should individuals within the community take to maintain their health?"; responses to this question are given in *Table III*. A strong emphasis on preventive medicine is evident, including such highly publicized measures as control of weight and diet, smoking, exercise, and alcohol or drugs. According to these data, students had well-formed ideas about health maintenance before the preceptorship, with particular concentration on the first four measures. It is interesting to find confirmation of a great shift in the social emphasis of medicine over the last few decades; such traditional medical concerns as sanitation and hygiene are far down the list, and perhaps are almost taken for granted, whereas the new concern for "life-style" problems is much more prominent. The most notable shift during the rotation (15-33% of mentions) was in compliance and follow-up by patients, perhaps as a result of exposure to patient apathy or fear in actual practice.

Students were also asked to list important primary health service supports within the community, and these responses are summarized in *Table IV*. Again, student concepts about health service supports were well-formed before the preceptorship, but it is notable that some supports taken for granted at a large urban teaching hospital such as UKSM receive a stronger emphasis after the rotation. For example, more emphasis was given to the availability of x-ray services, and a number of students noted the importance of ambulance services — perhaps because of the distances and transportation problems in rural areas. Nursing home facilities were strongly emphasized in post-test results, due to the number of geriatric patients in some small towns, and also be-

TABLE IV
STUDENT PERCEPTIONS OF PRIMARY HEALTH SERVICE SUPPORTS
n = 588

	Pre	%	*Post*	%
1. Hospital, emergency room, intensive care unit	294	(50.0)	286	(48.6)
2. M.D. specialists, consultations	205	(34.9)	162	(27.6)
3. Nursing staff services	197	(33.5)	132	(22.4)
4. State, county health departments	159	(27.0)	173	(29.4)
5. Laboratory, laboratory technicians	145	(24.7)	138	(23.5)
6. Social services, welfare	144	(24.5)	94	(16.0)
7. Allied health personnel	133	(22.6)	102	(17.3)
8. Clinics	121	(20.6)	90	(15.3)
9. Visiting nurses association	107	(18.2)	77	(13.1)
10. X-Ray, x-ray technicians	89	(15.1)	111	(18.9)
11. Pharmacy	86	(14.6)	71	(12.1)
12. Nursing homes	80	(13.6)	148	(25.2)
13. Ambulance	63	(10.7)	107	(18.2)
14 Voluntary agencies	62	(10.5)	41	(7.0)
15. Schools	31	(5.2)	41	(7.0)

TABLE V
STUDENT PERCEPTIONS OF BARRIERS TO DELIVERY OF HEALTH CARE
n = 588

	Pre %	*Post* %
1. Ignorance, fear	511 (86.9)	257 (43.7)
2. Cost	315 (53.6)	190 (32.3)
3. Lack of facilities	223 (38.0)	156 (27.0)
4. Lack of M.D.s, specialists	190 (32.3)	225 (38.3)
5. Accessibility	184 (31.3)	190 (32.3)
6. Availability	167 (28.4)	35 (6.0)
7. Lack of support personnel	146 (24.8)	82 (13.9)
8. lack of tests	80 (13.6)	41 (7.0)
9. M.D. lack of knowledge	39 (6.6)	33 (5.6)
10. Local competition	10 (1.7)	48 (8.2)

cause the students were exposed to the over-all scope of the health care delivery system, rather than only the large hospital. Schools received only a very small number of mentions, although they slightly increased in the post-test.

A corollary question concerns barriers to the delivery of health care observed in the community, as reported in *Table V*. Students tended to list fewer barriers after the preceptorship experience; some, for example, either left the space blank, or wrote: "None observed." Categories in which mentions increased were accessibility and lack of physicians/specialists, both problems associated with isolated rural practices. Only 5-6 per cent of the students noted what they felt was a lack of physician knowledge, although 48 noticed problems of local competition where only 10 had anticipated this.

The Preceptorship Experience

Student comments concerning the positive aspects of the preceptorship program have been tabulated according to a list of categories compiled from their responses *(Table VI)*. The items that appeared most frequently as positive features were exposure to private practice and exposure to medicine as practiced in the "real world" — common problems, procedures, and limitations. These comments indicate perhaps the most important educational aspect of the

TABLE VI
POSITIVE FEATURES OF THE PRECEPTORSHIP
n = 588

	Pre %	*Post* %
1. Exposure to "real world"	334 (56.8)	265 (45.1)
2. Medicine in a small town	183 (31.1)	163 (27.7)
3. Learning experience	177 (30.1)	159 (27.0)
4. See M.D. lifestyle	139 (23.6)	103 (17.5)
5. See common medical problems	108 (18.4)	60 (10.2)
6. More responsibility	107 (18.2)	131 (22.3)
7. Business, management aspects	91 (15.5)	112 (19.0)
8. Relationship with patients	86 (14.6)	54 (9.2)
9. Integrate skills	84 (14.3)	69 (11.7)
10. Small town environment	79 (13.4)	86 (14.6)
11. Get away from UKSM	63 (10.7)	63 (10.7)
12. Career decision	61 (10.4)	39 (6.6)
13. Gain confidence	24 (4.1)	99 (16.8)
14. Enjoyable	12 (2.0)	64 (10.9)
15. Preceptor himself	6 (1.0)	66 (11.2)
16. Staff	3 (0.5)	46 (7.8)
17. Other	106 (18.0)	68 (11.6)
Total	1663	1647

TABLE VI
NEGATIVE FEATURES OF THE PRECEPTORSHIP
n = 588

	Pre	%	*Post*	%
1. Separation from family, home	91	(15.5)	63	(10.7)
2. Isolation from social life	46	(7.8)	33	(5.6)
3. Lack of facilities, consultations	24	(4.1)	20	(3.4)
4. Long hours	18	(3.1)	20	(3.4)
5. Preceptor himself	15	(2.6)	8	(1.4)
6. Distance to travel	14	(2.4)	7	(1.2)
7. Living facilities	12	(2.0)	11	(1.9)
8. Specialty not available	11	(1.9)	7	(1.2)
9. Lack of supervision	8	(1.4)	11	(1.9)
10. Unbalanced patient population	7	(1.2)	12	(2.0)
11. Lack of clinical experience	7	(1.2)	4	(0.7)
12. Less than acceptable care	5	(0.9)	5	(0.9)
13. Interference with other electives	5	(0.9)	2	(0.3)
14. Attitude toward students	4	(0.7)	3	(0.5)
15. Other	82	(13.9)	82	(13.9)
Total	349		288	

entire program. It provides the only real exposure for many students to the type of medicine they will ultimately be practicing. The medical problems to which students are exposed at a tertiary care center such as UKSM are the statistically rare disorders which present in only a very small number of patients seeking medical care. Furthermore, in the post-test questionnaire, students observed favorably the friendliness of the small town environment, the staff and the preceptor himself, and the over-all enjoyable quality of the experience.

Exposure to rural practice, office management, and involvement with the preceptor's life style were also considered major benefits. The preceptorship was considered by a large number of students to be a positive learning experience and an opportunity to test their wings. They appreciated the responsibilities they were given for patient care which gave them confidence in their own capabilities as physicians and made them aware of their deficiencies and limitations. Students tended to have high expectations prior to the preceptorship, and most of these were met. The total numbers of positive responses concerning the program support the value of the experience.

This is even more significant when compared with the negative comments on the rotation *(Table VII)*. Some of these responses are probably unavoidable, especially that listed most frequently — separation from home and family. More and more students are married, and often have children as well. Many preceptors — especially in the regionalized clinics, groups, and hospitals — are making arrangements for spouse and family to accompany the student. For the most part, expectations of negative features were somewhat greater than those actually experienced. Post-test positive features outnumber the negative features almost 6:1.

In order to utilize the questionnaires for program development, students were asked to make suggestions for improvements in the program *(Table VIII)*. One-sixth of the students stated definitely "none" or that it had been an over-all good experience. Other

TABLE VIII
SUGGESTED IMPROVEMENTS
n = 588

	No.	%
1. None; excellent experience	105	(17.9)
2. More responsibility	60	(10.2)
3. More variety	62	(10.5)
4. Longer duration	62	(10.5)
5. Nearer home	43	(7.3)
6. Fewer hours or cases	28	(4.8)
7. Different living facilities	21	(3.6)
8. More instruction, feedback	18	(3.1)
9. Clearer roles, objectives	14	(2.4)
10. Another preceptor	13	(2.2)
11. Another location	13	(2.2)
12. More patients	11	(1.9)
13. Another specialty	10	(1.7)
14. Better hospital, facilities	8	(1.4)
15. Earlier in curriculum	6	(1.0)
16. Shorter duration	4	(0.7)

TABLE IX
MEAN STUDENT RESPONSES IN ESTIMATING THE PERCENTAGE OF OTHER CLINICAL ROTATIONS SURPASSED BY PERCEPTORSHIP IN TERMS OF CATEGORIES LISTED IN LEFT HAND COLUMN, 1976-1979

	1976 n = 138	*1977* n = 119	*1978* n = 164	*1979* n =119
Cognitive knowledge gain	43.4	46.0	49.1	50.3
Techniques	44.5	49.6	50.4	48.2
Enjoyment	70.8	73.9	78.2	73.8
Satisfaction	68.5	72.9	74.6	72.1

significant categories for improvement include more responsibility, and a wider variety of patients or cases. Suggestions for change have been relatively few and minor. They did not reflect problems with the time spent as much as the desire to be closer to home and family, or for extending the program.

Finally, students were asked what percentage of their other clinical rotations their preceptorship surpassed in terms of cognitive knowledge gain, psychomotor skills or techniques, enjoyment, and satisfaction. These results are presented in *Table IX*. It must be noted that most other rotations lasted eight weeks while the preceptorship lasted only four. Students have felt that the preceptorship was more beneficial than almost 50 per cent of the other rotations in terms of cognitive knowledge gain and techniques. There is some evidence of an improving trend in cognitive knowledge gain during the four years. This is significant in view of the fact that the preceptors are practicing physicians with busy schedules, little leisure time, and are not professional teachers or academics. Neither do they have, for the most part, the facilities readily available at UKSM. But the most noticeable result is the high level of enjoyment and satisfaction; the students evaluated the program as more beneficial than almost 75 per cent of the other rotations.

Conclusions

Our survey of the preceptorship indicates the value of the rotation to the student — academically both in terms of his degree of responsibility and the development of his medical skills, socially in his perceptions of the challenges and problems of community health, and in enjoyment and satisfaction. In the estimation of students during the past four years, this rotation compares very favorably with their experience at UKSM and in certain respects seems to surpass it. It may well be that this exposure to the traditional values of a small town experience and a very traditional kind of medicine in this country's history confirms to the student the value of a social and humanist content of his education and his practice as a physician. Furthermore, the formation of autonomous regional groups of physicians has provided a source of ideas not only for this rotation, but for the content of medical education. Finally, the preceptorship has assembled physicians, students, medical educators, and community leaders so that they may work practically together to address the economic, political, social, and organizational problems that the cost, quality, and delivery of health care present to our people and our nation.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Upper Gastrointestinal Bleeding: A Common Medical Emergency

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UPPER GASTROINTESTINAL (UGI) bleeding represents one of the more spectacular medical emergencies. It is frequently encountered in the medical practice of both the general practitioner and the consulting internist. Hematemesis induces anxiety in the patient and the physician, which improves only after stabilizing the hemodynamic parameters, stopping the bleeding, and establishing the diagnosis.[1, 2]

Causes

Several possible causes of blood loss from the UGI tract are listed in *Table I*. The most common are: (1) duodenal ulcer; (2) gastric ulcer; (3) gastritis; (4) esophageal varices; and (5) Mallory-Weiss (gastroesophageal junction) tears. Depending upon the patient population, these five lesions are responsible for 71-93 per cent of UGI bleeding.[1] Less frequently, anastomatic ulcers, esophagitis, and gastric neoplasms cause massive hemorrhage. Rarely, erosion of an aortic aneurysm or vessel graft into the upper tract results in rapid exsanguination. A family history of disease associated with UGI bleeding, such as the tumors of the multiple endocrine adenomatosis syndrome I (MEA-I) or mucocutaneous syndromes may lead the physician to the cause of bleeding. Patients with Zollinger-Ellison syndrome (gastrinoma) may have a family history of one or more neoplasms found in the MEA-I syndrome (parathyroid, pituitary, pancreatic). Mucocutaneous disorders (pseudoxanthoma elasticum, Ehlers-Danlos, hereditary hemorrhagic telangiectasia) are heritable and may be associated with mucosal vascular lesions of the upper tract. Lastly, idiopathic acquired vascular ectasias are unusual causes of upper bleeding.[3]

The frequency with which any lesion is found as the source of UGI bleeding is affected by multiple variables such as general health, age, alcohol consumption, drugs, and tumors. However, published studies suggest some generalizations. The differentiation between primary bleeding (causing hospitalization) and secondary UGI bleeding (developing in a patient hospitalized for another reason) is very important. In both groups of patients ulcer

TABLE I
CAUSES OF UPPER GASTROINTESTINAL BLEEDING

Most Common:	Duodenal ulcer
	Gastric ulcer
	Gastritis
	Esophageal varices
	Mallory-Weiss tears of esophagus
Less Common:	Esophagitis
	Gastric malignancy: primary or metastatic
	Anastomotic ulcers
Rare:	Mucocutaneous syndromes
	Erosion of aortic aneurysm or graft into GI tract
	Vascular ectasia

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(duodenal or gastric) is the most common lesion; however, in the group of secondary UGI bleeders the incidence of the hemorrhage arising from the mucosal lesions of esophagitis and gastritis is four times that found in the primary group.[1] In addition, many secondarily bleeding patients found to have an ulcer as the source of their bleeding, actually have a hospital-related stress ulceration.

Traditionally, hypotension and sepsis seem to have been accepted as the most important risk factors for stress ulceration and UGI hemorrhage among the critically ill. Renal failure and jaundice were the risk factors associated with the greatest incidence of acute UGI bleeding in patients already hospitalized.[4]

Probably the single most important factor in differential diagnosis of upper tract bleeding is alcohol consumption. In a report by Graham and Davis,[1] patients considered heavy alcohol users (>4 oz/day) had esophageal varices and mucosal tears as the most common sites of bleeding (58%). Sixty per cent of esophageal mucosal (Mallory-Weiss) tears with major bleeding occurred in patients with heavy ethanol ingestion. Acute ulceration — gastric and duodenal — was the most prevalent lesion (65%) among the nondrinkers.

Among patients with cancer, the most common cause of UGI hemorrhage is gastritis. This may occur secondary to stress ulceration or from gastric mucosal irritants such as analgesics or cytotoxic agents. Less frequently the tumor itself may be the source of bleeding. Massive hemorrhage secondary to the tumor mass itself is most commonly seen in primary gastric lymphoma or adenocarcinoma, although direct extension of a pancreatic neoplasm into the duodenum or stomach may occur. Finally, bleeding may result from metastases to the stomach. Breast and testicular cancers and malignant melanoma have the highest incidence of direct metastases to the stomach and small intestine.[5]

Initial Evaluation

Initially, the presence of blood in the UGI tract must be established. The history is probably the most important means of establishing the diagnosis of UGI bleeding. Patients with UGI bleeding usually present with hematemesis, melena or hematochezia, although syncope and orthostatic dizziness may be the only symptoms early in the course. Hematemesis and melena usually localize the bleeding proximal to the ligament of Treitz. Occasionally, however, melena occurs with the bleeding site in the small bowel or right colon. An UGI lesion with rapid, massive blood loss may present as hematochezia alone without hematemesis or melena and with a negative nasogastric aspirate. Black stools do *not* constitute melena; oral iron tablets and bismuth-containing compounds can cause black stools without the presence of blood. Likewise, ingestion of beets — and in the pediatric population, carrots and tomatoes — can cause red stools and be interpreted as rectal bleeding. Once the possibility of blood in the UGI tract has been established, bleeding from the bronchopulmonary tree (hemoptysis) and the posterior nasopharynx (epistaxis) with subsequent ingestion into the UGI tract must be ruled out.

After localizing the primary site of active blood loss, precipitating factors which may be associated with UGI bleeding should be considered. Several drugs have been implicated in upper tract hemorrhage, although only aspirin and alcohol are definitively associated with UGI bleeding. Acute alcohol use is associated with gastritis while chronic ingestion may lead to gastritis, ulcers, or bleeding varices. Chronic aspirin use (15 gm/wk) has been implicated in gastric ulceration and bleeding. Corticosteroids and non-steroidal anti-inflammatory agents have not definitely been found to be ulcerogenic, but may play some part in the pathogenesis of UGI bleeding in certain cases. Clearly the use of anti-coagulants such as heparin or coumadin is important historical information.

Reviewing patient symptoms prior to the onset of bleeding may provide useful clues. In those patients with symptoms indicative of ulcer disease and no previous abdominal surgery, 90 per cent were found to be bleeding from ulcers. In one series, an absence of a history of symptoms compatible with ulcer disease did not rule out ulcers as the cause of bleeding. In fact, only 43 per cent of patients with ulcers and massive UGI bleeding actually had classic symptoms of ulcer disease.[1] Therefore, a history compatible with ulcer disease is quite helpful but its absence does not exclude ulcers as the cause of bleeding.

Physical examination should first determine the extent of blood loss and secondly the site of bleeding. The most helpful physical finding in determining the severity of the hemorrhage is orthostatic change in the pulse and blood pressure. An estimated intravascular volume loss of 15-20 per cent can be estimated when there is a 10 mm Hg decrease in systolic pressure or a heart rate increase of 20 beats/min after the patient moves from the supine position to a sitting or standing position for two to three minutes. This suggests blood loss has been rapid and substantial and replacement of the intravascular volume is urgent. The physician should then look for clues that may be helpful in establishing the site of hemorrhage. Signs of liver disease — such

as jaundice, gynecomastia, spider angiomata, and hepatosplenomegaly — may suggest portal hypertension and possible variceal bleeding, although many patients with known esophageal and gastric varices bleed from another lesion. Signs suggestive of a mucocutaneous syndrome indicate one of these genetic diseases may be the cause of the hemorrhage. Petechiae and ecchymoses should be noted, as they point to an underlying coagulation abnormality or blood dyscrasia. Lastly, melanoma or carcinoma of the breast, lung, or testes should be sought.

Blood is found in the UGI tract by testing stool, vomitus, and nasogastric aspirate for occult blood. If a patient presents with hematochezia or melena and orthostatic changes, a gastric aspirate must be examined for gross or occult blood, though negative gastric aspirate does not rule out an UGI lesion as the bleeding source. In a series of 60 patients with massive UGI bleeding and negative gastric aspirates, 15 per cent bled from duodenal ulcers.[6]

Hints as to the duration and extent of bleeding can be found in the blood count and smear. A hypochromic, microcytic red cell appearance suggests a chronic problem while a normal blood smear and hemoglobin concentration in the presence of unstable hemodynamics is consistent with an acute bleeding episode prior to compensatory hemodilution. The immediate hematologic response is a leukocytosis as high as 20,000 followed by an elevation in the platelet count. The elevated reticulocyte count occurs later. A high BUN reflects the extent of bleeding and is elevated for two reasons: (1) blood degradation products in the intestinal tract; and (2) prerenal azotemia associated with hypovolemia. In the cirrhotic patient, the BUN elevation may be the harbinger of hepatic encephalopathy. Coagulation parameters may reflect the cause of bleeding and are critical to appropriate management.

Definitive Evaluation

The bleeding site may be identified by endoscopy, barium studies, or arteriography. A comparison of these diagnostic approaches is found in *Table II*. Considerable controversy has evolved regarding the value of endoscopy in UGI bleeding. Most individuals agree upper endoscopy is an excellent diagnostic tool when performed by properly trained personnel. In most series, upper endoscopy can determine the bleeding site in about 90 per cent of cases and localize the bleeding to a particular organ in up to 95 per cent. It has the advantage of diagnosing mucosal lesions and identifying the bleeding source when multiple possibilities exist. Controversy surrounds the questions of when endoscopy should be done and whether endoscopy benefits the patient. Several investigators have shown that endoscopy does not decrease mortality or duration of hospital stay and is not mandatory for the proper care of all UGI bleeders.[7] Endoscopy should be done by those properly trained in the procedure as the complications of aspiration pneumonia, cardiac arrest, perforation of a hollow viscus, or massive iatrogenic bleeding are negligible in the hands of a competent endoscopist, but occur too frequently among the untrained.

Radiological studies are often helpful in determining the site of bleeding. Radiographs are most effective in detecting ulcer disease, esophageal varices, and tumors. They cannot distinguish mucosal abnormalities such as gastritis, esophagitis, or Mallory-Weiss tears of the esophagus.[7] In addition, when several lesions are present concurrently, barium studies are unable to detect which lesion is

TABLE II
DIAGNOSTIC EVALUATION OF UGI BLEEDING

Procedure	*Indications*	*Advantages*	*Disadvantages*
Endoscopy	Stabilizing UGI bleeding	Rapid, precise diagnosis	Trained personnel; less effective in massive bleeding
Barium studies	Ulcer suspected; endoscopy not available	Less invasive; effective in diagnosis of ulcers	Misses mucosal lesions; not effective in massive bleeding
Arteriography	Massive bleeding	Localize site in massive bleed; modality of therapy	Requires trained personnel and active bleeding

bleeding. Despite these shortcomings, if endoscopy is not readily available, contrast studies should be performed, provided the patient is stable and not actively bleeding. A barium test should not be performed on any patient with a suspected perforation, or in whom arteriography is considered in the near future. If bleeding is massive and continuous, the diagnostic procedure of choice is mesenteric arteriography. Continuous, profuse bleeding greater than 1 ml/min renders endoscopy less fruitful while increasing the diagnostic yield of arteriography. It frequently pinpoints the site of bleeding and provides the physician with another modality of medical treatment; vasoconstricting or thrombosing substances may be introduced via the mesenteric catheter to control the bleeding.

Management

The cornerstone of medical treatment of UGI bleeding is expansion of the vascular space. After a physical examination, during which the stability of the intravascular volume has been estimated, fluid replacement should be initiated with an isotonic crystalloid solution. If orthostatic changes of pulse or blood pressure are found, a solution of normal saline or Ringer's lactate must be started to maintain vascular volume until whole blood is available for transfusion. It is important to emphasize the use of isotonic solutions in this setting. Only 12 per cent of hypotonic fluids such as 5 per cent dextrose in water is maintained in the intravascular space. Therefore hypotonic solutions would not adequately maintain vascular volume in this life-threatening setting. Usually replacement can be adjusted while monitoring pulse, blood pressure, and tissue turgor; however, evaluation of central venous pressure with an intravenous device may be necessary.

Several modalities have been used in an attempt to terminate UGI hemorrhage. Lavage of the upper tract with ice water or saline is a time-honored tradition. This procedure, using a large lumen nasogastric tube, usually adequately removes clots from the stomach in anticipation of endoscopy or roentgenographic studies. The efficacy of lavage in actually terminating the bleeding event has not been documented. If ulcer disease or gastritis is the suspected cause, adequate doses of antacids or cimetidine may be helpful. Many years ago it was discovered that the vasoconstricting agent vasopressin is helpful in controlling an acute UGI bleeding episode, especially when the cause is esophageal varices. Recently the intravenous route has been found just as effective as intra-arterial infusions in the patient bleeding from esophageal varices. A useful regimen for vasopressin begins with 0.4 units/min until bleeding stops or 24 hrs. After control of bleeding is achieved, the dose is tapered to 0.2 units/min for 24 hrs, then 0.1 unit/min for 24 hrs, then saline for 24 hrs. If rebleeding occurs, the initial dose is reinstituted.[8] It must be emphasized that although vasopressin may shorten the bleeding episode in these patients, it does not increase the survival rate. If massive hemorrhage continues in spite of adequate volume replacement and appropriate drug therapy, surgical intervention is necessary. Minimally invasive procedures — such as electro and laser coagulation of mucosal lesions and injection of sclerosing and thrombosing agents into esophageal varices — will be available in the future.

In summary, UGI bleeding is a serious, life-threatening catastrophe that is appropriately handled by stabilizing the patient, then analyzing the history and physical examination to determine the site and cause of hemorrhage. Gastric and duodenal ulcers, gastritis, esophageal varices, and Mallory-Weiss tears of the esophagus are the most common causes. Endoscopy, barium studies, and arteriography usually reveal the site of bleeding. Vigorous fluid replacement, appropriate drug management, and surgical intervention are the modalities that will control most bleeding episodes.

Self-Assessment Questions

1. The single most important historical factor in the differential diagnosis of upper gastrointestinal bleeding is:
 a. family history of similar problems
 b. glucocorticoids use
 c. alcohol intake
 d. concomitant neoplasm
 e. aspirin use
2. Important risk factors for stress ulceration are:
 a. sepsis
 b. renal failure
 c. hypotension
 d. jaundice
 e. all of the above
3. The most common cause for bleeding in the cancer patient is:
 a. tumor metastasis (breast, testicular, melanoma)
 b. gastritis
 c. primary tumor (gastric, lymphoma)
 d. esophageal tear (Mallory-Weiss)
4. A 10 mm Hg decrease in systolic pressure or an increase of 20 beats/min in the pulse rate when the patient moves from the supine position to the

sitting or standing position represents a loss of intravascular volume of:
 a. <5%
 b. 5-15%
 c. 15-20%
 d. 20-35%
 e. >35%
5. Endoscopy decreases both the mortality and hospital stay of patients with upper gastrointestinal bleeding. (T or F)
6. In patients with bleeding esophageal varices, intravenous vasopressin is as effective as selective intraarterial vasopressin. (T or F)

(Answers on page 155)

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Fetal Cardiac Arrhythmias

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Puzzling Liver Nodules

(Continued from page 133)

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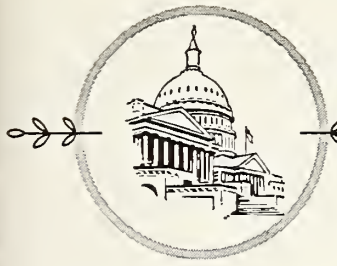
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Socio- ECONOMICS

The Importance of Checking References

MOST PHYSICIANS will not experience dishonesty in a medical assistant, but an incompetent or ill-mannered employee may be just as great a liability. The best indicator of future success is past success — for everyone in the practice from physician to file clerk. The possibility for making the "perfect hire" can be enhanced by checking the past performance of all potential employees.

Unfortunately, many physicians do not check references — and many of them get burned as a result. One doctor hired a new assistant on the recommendation of a past employee who had known the woman. She was new in town and was anxious to find a job. He needed someone in a hurry and trusted the word of the former assistant. The applicant was hired the same day she was interviewed, and when she reported for work, the doctor was dismayed to find that she was unqualified as a typist and short-tempered with patients. A long-distance call to her previous employer might have saved much expense and grief.

Two points worth keeping in mind when checking the references of job applicants are: (1) "personal" references may be of little value because these persons have been carefully chosen to give nothing but good reports; and (2) permission from applicants is necessary prior to contacting former or current employers. Such approval may be a part of a job application form. An applicant who refuses such permission should give reasons. Often an applicant's current employer is unaware that he/she is seeking another job, and if that is the case, the final decision on employment should be contingent on that additional reference. One office manager decided against hiring an otherwise acceptable insurance clerk when the final reference check revealed prior termination for excessive tardiness and absenteeism.

Gaps in past employment history should be checked also. An applicant may have spent two years at home raising a family; or he/she may have skipped from job to job, lasting only a few months at each.

In contacting prior employers, it is best to use the telephone, as many people are hesitant to put negative reports in writing. It is better to speak with the applicant's past immediate supervisor — rather than the personnel department — for information from someone who worked in direct contact will be of greater value.

In such conversations, it is important to listen to what is *not* said as well as what is said. It is rare that anyone will make direct negative comments; however, they will often imply them by weak responses to questions.

Personnel experts caution against asking discriminatory questions when checking references; queries regarding an applicant's religion, race, or family plans are just as inappropriate in this context as in interviewing or on an application, and only information relating to the applicant's ability to perform the job may be solicited.

One question that should be asked of all previous employers is whether they would be willing to rehire the person. An immediate "yes" speaks well for an applicant; a flat "no" or even a moment of hesitation may suggest the desirability of reconsideration.

Many past employers have strict policies about information given by telephone. Some will only reveal how long the person was employed; others will require that all reference checks be submitted in writing. Perhaps a past employer will only verify information already provided by the applicant, but this may be sufficient to confirm that the applicant is telling the truth about past length of service, job responsibilities, and salary level.

In a recent Texas court decision, a patient injured

(Continued on page 154)

From AMA Department of Practice Management, Division of Medical Practice, Chicago.

The President's Message

Influence

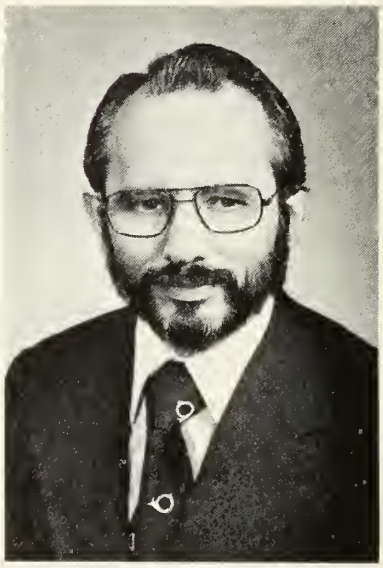
Each of us is a product of our heredity plus our environment. Our heredity is predetermined and therefore not something that we can change. Our environment, however, while unique for each of us, is at least partly under our own control.

Many of us are in medicine because of the influence of our environment. It may have been the personal influence of a parent, teacher, friend, or perhaps a physician. The influence may have been from a personal bout with illness or injury, or the illness or injury of a family member or a friend.

Much of our environment is not under our own control. A mother may project the desire for her child to be educated because she herself was unable to get an education; or a parent may project a very definite feeling that education is a waste of time. Early in our lives we may have had a teacher who convinced us that the pursuit of knowledge was a worthy goal, thereby instilling within us a burning desire to obtain an education. It is amazing how the influence of one teacher can ignite the spark of intellectual curiosity in a student.

But part of our environment is under our own control. We can choose to work, loaf, play, read, think, etc. We determine how we spend our time. Our choice of friends and activities will affect our final character and personality. Each of us has had many positive and negative influences in our life; some we remember well and many others we have pushed back into our subconscious minds.

Each of us also exerts a daily influence on many people around us. For the most part these influences are casual and frequently mundane. Some seemingly very trivial comment or action occasionally has a tremendous influence on another person's whole life. I am constantly amazed at the number of times that a casual remark or action on my part has made a deep impression on someone. In a similar fashion, some of my most concerted efforts to influence others have seemingly had no effect on anyone.



As physicians we hold a position of fairly high esteem within our communities. We are looked up to as leaders in the community and, therefore, our comments and actions may be more closely scrutinized than those of someone who is not in that position of leadership. Even trivial comments we make may have a profound positive or negative effect upon someone with whom we come in contact.

Our personal influence is important. Every contact we have with the public, with our employees, hospital personnel, the people in our communities can be either a positive or a negative influence. Remember, we do have more influence than the average person. It is up to us to see to it that this influence is a positive one.

Fraternally,

Donald D. Gerwig, MD

President



Editorial COMMENT

Rights Face

—Court action is brought to override the parents' decision to withhold life-prolonging surgery on an institutionalized mentally defective child.
—Court action is sought to permit (require?) the use of a therapy on a minor child contrary to the parents' religious or moral scruples.
—Legislative action is sought to establish permission to allow "natural" death by withholding sustaining treatment.
—Legislative action is sought to provide compensation of voluntcer subjects of human experimentation who suffer ill effect from it.

These generalized and somewhat disparate but not unfamiliar examples of the search for "rights" have appeared with increasing frequency in recent years. They point to a significant shift in medical responsibility which is both cause and effect — effect since they represent an altered medical function in a world reliant on legal precedent and cause since they seek actively to establish for future use a specification of medical behavior to relieve society of repeated confrontations with such problems. Mankind has known no greater stimulus to its search for answers than the desire to eliminate such distressing questions, that is, resolve them by applying a predetermined solution.

The extent of this country's change in social philosophy is probably greater than we realize since we have thinned it by comforting sematics and denial. We have prided ourselves on the stability of our government (which has, through times of great ferment, avoidcd a classical shooting revolution) and have come gradually to accept — expect? — things that were anathema not too long ago — medically as well as socially.

New techniques and new therapies have led to new conditions and new expectations. The physician of today is as different from his predecessors as are his methods from theirs, and by no means the least part of this change has come from the incursion of society into medical decisions — both personal and policy. Consumer advocates would have it that we are seeing the righteous and overdue reaction of an abused patient population demanding the return of its health franchise so crassly appropriated by a self-serving profession. As a matter of fact, the social actions couldn't have brought on these changes alone. The medical profession has helped by sometimes ignoring, sometimes abdicating, sometimes misreading when it found itself unprepared or unwanting of the task. If this sounds unduly critical, it is not so intended. It simply points out that physicians in a simpler (and medically less effective) time enjoyed an autonomy regarding their patients' medical problems, passing over the social implications. Their decisions sufficed since there were no other advocates or functional considerations than care of the patient.

It wasn't a worse system than the other elements of the social structure — probably somewhat better since it righted some wrongs and led more or less directly to the implementation of education and research and an improved quality of life which combined to foster the changing concept of medical service including the increased involvement of the public in medical matters — even unto the current manifestations.

But such exclusions from accountability as the physician formerly had has been consistently eroded since he has been admirably prepared for his medical function but scarcely at all for his social one. He has generally approached these sociomedical problems with one or the other attitude — a stubborn insistence on applying only a medical interpretation or an abdication in favor of the governmental, legal, or social entity which would (with little urging) have to meet the problem. In the process, he is left dismayed that he has lost title to some of his supposedly sacred functions and must negotiate in a hostile atmosphere to regain even a voice in such matters. He is learning

that if he resorts to asking the courts — or any other agency — to resolve his social dilemmas, the price for what he deems good medical practice is the transferral of some part of his professional control over to the social interlopers. The painful precedent becomes, unfortunately, progressively less difficult, then accepted, then the norm, then the demand.

In the current climate, the certain way to gain support for any effort is to insist on it as a "right" or proclaim that any denial constitutes a violation of a right. This makes for excellent rhetoric since the individual so confronted, being uncertain how the "right" should be implemented or protected (or if it even *is* a right) must call upon one or another of these agents for a decision.

All living matter is egocentric, a trait that increases as one ascends the phylogenetic scale with man being the most obvious example up to now. Each species has characteristic forms of expression, but man has refined his through the application of the "right" principle. This proclaims that in any one of his various social or ethnic or political or whatever forms, he is entitled to certain guarantees, not just for survival but for society to meet the stated specifications of those forms. But a right for any one person or group imposes restrictions on others or there would be no need to formulate them. Our considerable supply of undesirable characteristics provides ample material for the dedicated rightists. If these could be arranged into neatly interlocking and mutually supportive pieces of a die-cut jigsaw puzzle, it would be utopian, but instead we have the overlapping fragments of a kaleidoscopic cluster — of illusory attraction and subject to change with a twist of the social wrist. However desirable such rights may be (and God is usually cited as the originator of any particular right under consideration), the unlimited and piecemeal pronouncement of various rights threatens to create social, political, and moral congestion in which no rights are respected.

Parliamentary action doesn't have too good a showing in the rights area. True, our Bill of Rights has done well but suggests one should really get in on the ground floor to have much effect. The Emancipation Proclamation, as significant a piece of rights action as we have produced, has yet to achieve the social and moral intent. Currently, the ERA effort is looked upon by many as the Equal Wrongs Amendment. Insofar as there is a lesson in the efforts to legislate rights, it would seem that the mind can't do it if the heart isn't in it.

The rights concept is obviously a virtuous one but the element of virtue is malleable. At the moment, for example, the use of placebos, an essential mechanism for evaluation in research not to mention its value in utilizing the patient's psychological resources without medicinal harm, is under attack as violating the recipient's "right" to know what he is getting and why. The traditional position of the physician has been that he, being the medically informed and responsible party to the transaction, knew what should and should not be disclosed to the patient regarding his condition. This traditional function must now, however, be accomplished according to the dictates of the patient's "rights." The difference (if we can detect it) will apparently be one measure of the substance of those rights, and if there is still doubt, we can rely on court decisions or the directives of other agents to inform us. Ironically, the one major difference that emanates from this rights hyperactivity is — compulsion.

So the various rights movements seem to represent the growing pains of the civilizing process, and medical matters will continue to occupy a prominent place since medical service runs a close second to the basic survival necessities of life. Along with a few other perplexing matters, we have the task of assessing and developing the character and performance of our services so as to demonstrate that we recognize them and respect them *as* rights without the need for other agents to intervene. — *D.E.G.*

The Importance of Checking References

(Continued from page 151)

by an incompetent orderly successfully sued the hospital for punitive as well as compensatory damages. The hospital had not checked the orderly's references when they hired him. As a result of the suit, it was discovered that he had been expelled from corpsman's school and had a criminal record for drug violations. The court ruled, "Gross failure in making sure employees are qualified and trained for assigned tasks will subject a hospital to such damages." It is not stretching the imagination to assume that the same would hold true for a physician's office.

Because employee salaries are one of the largest costs of a practice, it is worth while to spend some time and effort in making the best possible hire.

Ka M P A C

KANSAS MEDICAL POLITICAL ACTION COMMITTEE

1300 TOPEKA AVE. • TOPEKA, KANSAS 66612

Dear Doctor:

How does the governmental bureaucracy view medicine?

With any given disease, all patients would have the same pain threshold, emotional response, and morbidity; all would respond similarly to the same treatment.

Doctors should have the same efficiency, speed, and philosophy of patient care; they should treat a specific disease in exactly the same standardized manner at all times.

Each disease would have its own code number, and each coded disease should produce identical symptoms in all patients; each disease would respond in the same manner in every patient using the standard coded treatment regimen.

Why do the bureaucrats view medicine in this manner? By this process medicine can operate in quantitative rather than qualitative methods. Information can be fed into a computer for analysis by someone with little knowledge or training.

KaMPAC wants to preserve the qualitative aspects of medicine — its humanism, flexibility, responsiveness, and its view of the patient as an individual.

Your generous contribution to KaMPAC can help the medical profession preserve its ability to provide excellent health care.

Sincerely,

Ronald Davis, M.D.
Chairman

Dramamine in Labor

(Continued from page 136)

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Upper Gastrointestinal Bleeding

(Continued from page 150)

Answers

1. *c*
2. *e*
3. *b*
4. *c*
5. *False*
6. *True*

Rambling Recollections . . .

(Continued from page 102)

misused, and it was probably actually to the financial advantage of the school to manage the money in that manner.

Among the first programs to be organized were the Circuit Courses which were designed to bring medical education to the physicians in small rural communities throughout Kansas, and for this purpose two circuits were organized: Western and Eastern with four towns each. In that way eight different towns throughout the state received six programs annually. The programs were scheduled to start at 2:00 p.m. There was a recess for dinner with the faculty and enrollees eating together, and the program then resumed to continue until 9:00 p.m.

Two staff members were needed to take the teams out, so Mr. Ingham elected to tour the Western Circuit while the registrar of the School of Medicine, Mr. Herbert Weatherby, escorted the eastern team. A field representative and four faculty members comprised each team, and each member usually gave two talks. There were arrangements for panel discussions, and the last item on the program was a "wet clinic" to which local doctors were invited to bring problem patients for members of the faculty team to comment upon.

The programs were planned by the Joint Advisory Committee on Postgraduate Education, which also chose the towns in which the Circuit Courses would be offered. It was my privilege to attend these meetings beginning in 1953. The decision about which towns to visit caused a certain amount of controversy and took a great deal of time. Rather keen competition existed between several towns in contiguous or nearby counties and local pride did not permit an easy surrender of the program to another competing town. The Advisory Committee also spent a great deal of time outlining precisely how each program would be carried out both as to which faculty members should be asked (or required) to go and what their topics would be. At that time it was the policy of the school to indicate to all full time faculty members that they were expected to "go on the Circuit" if they were needed. In addition to local faculty they also often asked some specialist from Topeka, Kansas City, or Wichita to accompany the team as a full-fledged member. It was common for the annual Advisory Committee meetings to last as long as 12 hours with recesses for luncheon and dinner.

In the early days of the Department of Postgraduate Medical Education there were only a few

(Continued on page 157)

Tenuate®
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema; *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico 00633

Direct Medical Inquiries to
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

Licensor of Merrell®

References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.: A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404

Merrell

9 4672 (Y957A)

Overweight may not always be simple... complications can develop.*
Complicated or not...

Tenuate® Dospan® ℂ℣
(diethylpropion hydrochloride NF)
75 mg. controlled-release tablets



A useful short-term adjunct in an indicated weight loss program.

Overweight patients in certain diagnostic categories often require strict appetite control and a successful program of weight reduction may tend to diminish the incidence or severity of the complications in some patients. Diethylpropion hydrochloride has been reported useful in such patients and while it is not suggested that Tenuate itself in any way reduces the complications of overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. **Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on the opposite page.**

In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness.

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

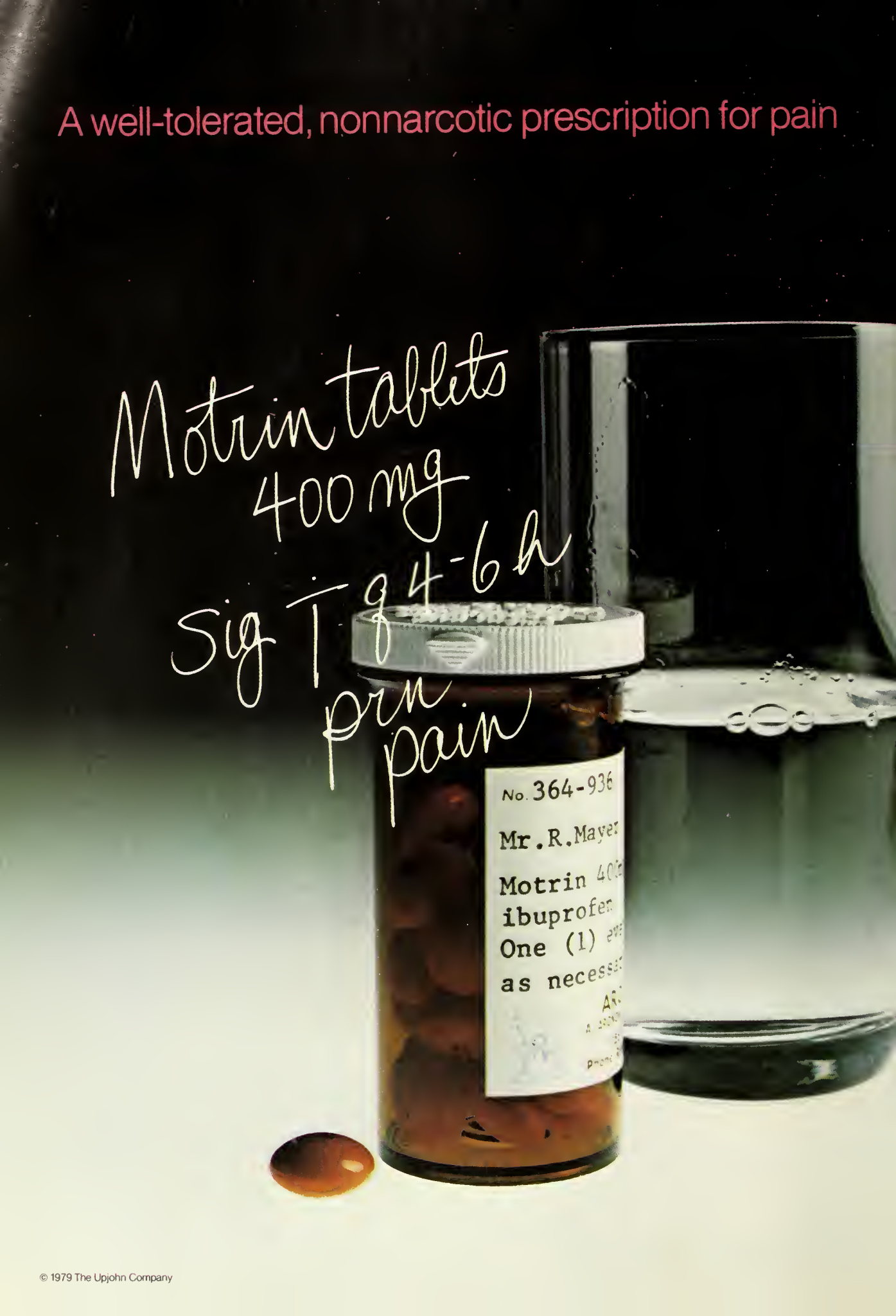
Tenuate—it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

Merrell

For prescribing information see opposite page.

A well-tolerated, nonnarcotic prescription for pain



© 1979 The Upjohn Company

Motrin® now proved an effective analgesic for mild to moderate pain

Motrin 400 mg provided greater relief of pain than did propoxyphene 65 mg in controlled clinical pain studies.

Time after drug administration (hour)		.5	1	2	3	4
Mean relief-of-pain scores* (No. patients reporting)	Motrin 400 mg ibuprofen	.89 (108)	1.25 (108)	1.36 (108)	1.28 (107)	1.19 (106)
	Darvon 65 mg propoxyphene	.66 (100)	.99 (99)	1.13 (96)	.99 (96)	.80 (96)
Statistical significance		$p<0.02$	$p<0.01$	$p<0.05$	$p<0.02$	$p<0.002$

*0 = No relief 1 = Partial relief 2 = Complete relief

Data on file at The Upjohn Company

Motrin demonstrated statistically significant greater relief of pain than did Darvon at all time intervals.

Motrin 400 mg TABLETS
ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming
- Rapid analgesic action • Indicated in acute and chronic pain
- Well tolerated. The most common side effect with Motrin is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen) now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.
Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

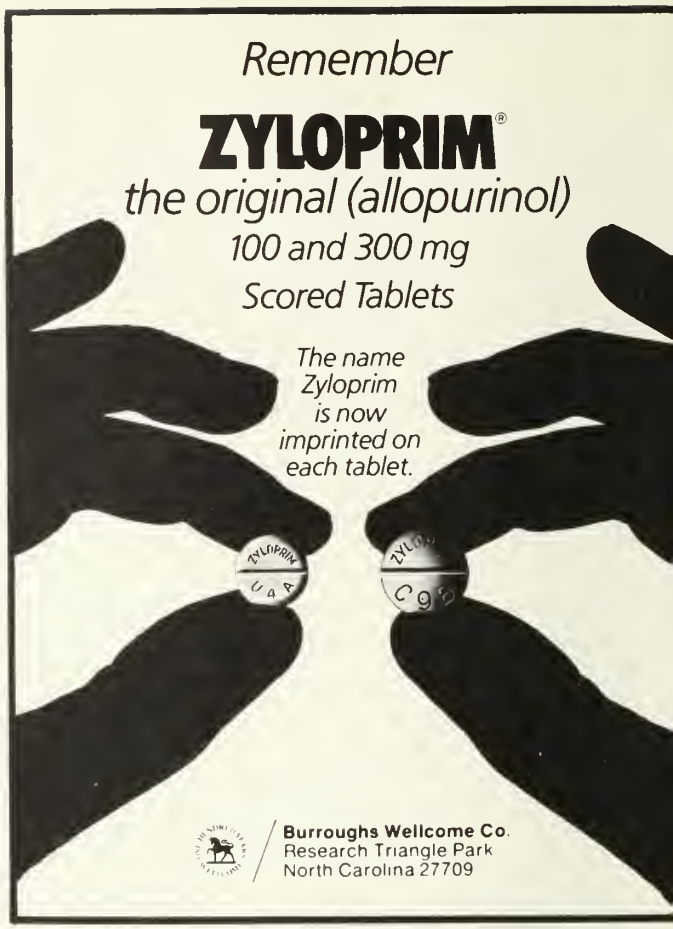
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Rambling Recollections . . .

(Continued from page 156)

major symposia, each of which enrolled from twenty-five to seventy-five physicians. The major effort of the department at that time was the Circuit Course which was part of the so-called "Murphy Plan." Among other things the Murphy Plan was attempting to get clinics built in small communities so that they could attract physicians — the idea being to replace the old timers who were retiring with solo practitioners in the really small towns. As mentioned above, there were no established centers for the Circuit Course, a new set of towns being selected each year, but that had to be revised so that there was some stability and enrollment began to increase as physicians began to look forward to attending a program in a certain place. This helped them form an "educational habit."

In addition to the Circuit Course there were symposia four or five times a year in the major disciples of medicine. At first these symposia were attended largely by specialists from Kansas and western Missouri, but as time went on they began to attract more highly motivated general practitioners who found that they were welcome. In 1950, Dr. Hashinger retired as chairman, and Dr. Delp became chairman of the Department of Postgraduate Medical Education in 1951 (in addition to being professor of medicine). Dr. Delp and Dr. Wescoe felt that the contact with general practitioners in the state should be increased and that the preceptorship program — which was part of Chancellor Franklin Murphy's "Kansas Plan" — should be somewhat fortified. Since I was in private general practice in Kansas City, Missouri, while retaining a teaching position in the Department of Pharmacology (which I had held since 1939) as well as a position of clinical instructor in the Department of Medicine, I was asked whether I would join the full-time faculty with joint responsibilities for pharmacology, medicine, and postgraduate medical education on July 1, 1953, an offer which I accepted.

My first contact with the Department of Postgraduate Medical Education had been through my relationship with the Missouri Academy of General Practice and its Kansas City Chapter. I had been chairman of the education committees of both chapters and as such had been instrumental in establishing a local course for general practitioners in the greater Kansas City area at the University of Kansas Medical Center. This program had been quite successful and brought me to the attention of the administration of the school. It also had given me a friendly working relationship with Mr. Ingham, which later proved to be a great advantage.

With Dr. Delp as chairman of the department, additional symposia were added to the program more rapidly than previously. The duties of the chairman were increasing rapidly, and it was my assignment to help him with some of them. Among other things, I became a member of the Advisory Committee and acted as its recorder. The Advisory Committee meetings became increasingly burdensome so Dr. Delp and I gradually began to press upon the committee the desirability of stability and location of the Circuit centers. We were never able to reach complete stability, but it was then that the Circuit Course began to take on its present shape.

Symposia at the Medical Center were offered in the auditorium of the Children's Pavillion, in the third floor clinic auditorium, and in the postgraduate auditorium which was on the fifth floor of the Clinic Building (now Sudler Hall). This auditorium had a capacity of about 75, and was ideal for presenting postgraduate courses in the earlier stages of development. As courses became more popular, attendance rose to the point where there was often standing room only. At about that time (1953) the Battenfeld Auditorium was completed as part of the Student Center-Continuation Study Building which was built to be the main locus for symposia. As the space increased and as numerous primary care physicians began to attend the specialty symposia, their enrollment increased, and more were offered.

Enrollment in the Circuit Course had been increasing in the meantime, and it was necessary to allow Mr. Herbert Weatherby — who had been acting as part-time field representative — to resume full-time duties as registrar, and a field representative was hired. Later, as Mr. Ingham neared retirement, it became necessary to hire a second full-time field representative so as to relieve Mr. Ingham of that duty. Mr. Ingham was a very economical person and drove a small car. His vehicle, a Studebaker Champion, was so light that when he drove at his usual speed (flat out) its wheels behaved something like the feet of a five-gaited horse. It was his habit to point out landmarks and tell stories about doctors as the teams drove along, and he habitually turned to look into the back seat to talk to the members of the faculty who were sitting there, using at least one hand to point to landmarks while assuming that the car would find its own way to its destination. From personal experience I am convinced that once he had attained cruising speed, the car never had more than one wheel on the ground at a time. Mr. Ingham knew every doctor in western Kansas (and probably in

eastern Kansas as well), and could tell many stories about them.

William Nelligan became available as a field representative in 1956 with the understanding that if his work proved to be satisfactory he would become executive director upon Mr. Ingham's retirement. When Mr. Nelligan became executive director in 1957, the staff consisted of Mr. Nelligan, Jane Jackson, and another secretary who had joined the group, as well as Dr. Delp and myself. Dr. Delp arranged — with the help from the money in the Kress Foundation which was given to the Department of Postgraduate Medical Education — to build office space in connection with the Student Center-Continuation Study Building. Until then the office of the executive director consisted of a cubbyhole outside of the postgraduate auditorium on the fifth floor and the even smaller office for Jane Jackson and the other secretary who had been hired to help with the work. These two women typed, stamped, and mailed all promotional brochures and other correspondence from the department. After the building was built adjoining the Battenfeld Auditorium, additional space became available to include added staff to take care of the increased enrollments. Mr. Nelligan introduced new business methods in promotion so that mailing lists were purchased, and a large part of the local addressing was accomplished with Addressograph plates. This allowed the department to publicize its courses more widely and enrollment increased rather rapidly. It was necessary to employ one clerk practically full-time to keep Addressograph plates in order and to perform other duties connected with expanded mailing lists.

We had also increased work in preparing course notebooks and managing enrollments so that the staff was enlarged to include a registrar and two clerks in addition to the administrative group (of which Ms Jackson became a *de facto* member) and the field representatives. The quarters of the department consisted of a registration area, an office for Ms Jackson and the field representatives, an office for the executive director, an office for the chairman, a conference room/classroom, and a workroom for storing material to go into course notebooks as well as for preparing those notebooks. The workroom rapidly became inadequate and was converted into office space while the classroom/conference room was converted into a workroom and the field representatives were ultimately moved out of Ms Jackson's office. In 1969 even this arrangement became overcrowded and, with its Kress Fund and Endowment money, the department purchased a "temporary building" which became a storage room and workroom, and was erected adjacent to the regular office space. The erstwhile workroom was converted into offices for the field representatives.

In 1960 Dr. Delp became chairman of the Department of Medicine and I became chairman of the Department of Postgraduate Medical Education, and in 1964 Mr. Nelligan was replaced by Robert L. Neth, Sr. The work of the "number one" secretary had become less secretarial and more managerial and the position was changed to assistant director so that the administrative staff of the department consisted of a chairman, executive director, and an assistant director.

In 1967 I was also named as associate director for the Kansas Regional Medical Program which was established with federal money. My duties were to head the continuing education portion of that program while being relieved of some teaching duties in pharmacology and medicine. One of the projects that we instituted was the Circuit Course for Nurses. This was a Regional Medical Program project which became self-supporting in a relatively short time. It was necessary in order to develop and maintain the Circuit Course for Nurses to add a third field representative, and Frank Tyler was recruited for that job (with the title of assistant director for outreach programs) thus increasing the administrative staff to four. The Circuit Course for Nurses was set up on much the same pattern as that for physicians with the exception that each center would have only four programs. In addition to this there would be a program in Kansas City for nurses in the northeast part of Kansas and in western Missouri.

The 60s and the 70s until about 1976 should probably be called the golden years in continuing education. In 1976 the era of purely voluntary continuing education was coming to an end with relicensure based on continuing education credits.

During those golden years this Division gained national prominence in continuing education because we conducted a variety of programs and staged them in a thoroughly professional manner with an efficient and effective staff. Our reputation brought 183 visitors exploring continuing education techniques from 30 states outside of Kansas and from 22 foreign countries.

As my retirement from this Department approaches I am turning over my duties to Dr. Joseph Meek, and the Division of Continuing Education is combining with the Division of Health Care Outreach.

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SECOND OPINION ON SURGERY

A nationwide information campaign to encourage people to get a second medical opinion before undergoing non-emergency surgery is about to begin, HEW announced today.

The campaign will include radio and television announcements featuring actor Cliff Robertson, brochures, posters and a toll-free telephone hot line through which patients can get the names of physicians willing to give second opinions.

Campaign announcements will also encourage patients to discuss frankly with their physicians how to obtain a second opinion for non-emergency surgery.

The campaign is being conducted by HEW's Health Care Financing Administration, which administers Medicare and Medicaid, and is directed to the general public and especially the 47 million Medicare and Medicaid beneficiaries.

HEW estimates that in 1976 more than 20 million operations were performed annually on in-patients in the nation's hospitals; also, that the surgery rate for persons aged 65 or older increased 44 per cent between 1966 and 1976.

First year results of a three-year demonstration project being conducted in New York for the Health Care Financing Administration under an HEW contract to Blue Cross/Blue Shield of Greater New York show that in 31 per cent of 1,464 cases recommended for surgery, the need for surgery was not confirmed by physician consultants giving second opinions. The first year of a three-year Michigan demonstration being conducted for HCFA under contract to Blue Cross/Blue Shield of Michigan showed similar results.

In the New York project second opinions have been made available without charge since May 1978, to more than a million Medicare beneficiaries in 17 counties of New York and all five New York City boroughs.

The Michigan project, underway since August 1978, covers more than 365,000 Medicare beneficiaries in Wayne, Oakland, and Macomb counties and the city of Detroit.

Medicare pays for second opinions for surgery nationwide, and Medicaid covers second opinions in most states.

HLA TESTING

HLA Testing for disease association studies, HLA B-27, HLA-B8, etc. Available from the Midwest Organ Bank Laboratories.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability, insomnia associated with anxiety neuroses and transient situational disturbances; an associated with depressive symptoms and as a treatment of symptoms of anxiety if such s toms are a significant feature of functional or organic disorders, particularly gastrointestin cardiovascular

Effectiveness in long-term use, i e , more than 4 months, has not been assessed by sys atic clinical studies Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucon

Warnings: Not recommended in primary depressive disorders or psychoses As with all (acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, a diminished tolerance for alcohol and other CNS depressants

Physical and Psychological Dependence Withdrawal symptoms like those noted with b turates and alcohol have occurred following abrupt discontinuance of benzodiaze (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). A tion-prone individuals, e g drug addicts and alcoholics, should be under careful surveill when on benzodiazepines because of their predisposition to habituation and depende Withdrawal symptoms have also been reported following abrupt discontinuance of ben azepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid sedation

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may resu symptoms like those being treated anxiety, agitation, irritability, tension, insomnia and o sional convulsions

Observe usual precautions with impaired renal or hepatic function

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that loraze has not been shown of significant benefit in treating gastrointestinal or cardiovascular cor nent

Esophageal dilation occurred in rats treated with lorazepam for more than 1 ye 6mg kg day No effect dose was 1 25mg kg day (approximately 6 times the maximum hu therapeutic dose of 10mg day) Effect was reversible only when treatment was withdrawn w 2 months of first observation Clinical significance is unknown, but use of lorazepam for longed periods and in geriatric patients requires caution and frequent monitoring for sympt of upper G I disease

Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia, some have elevations of LDH As with other benzodiazepines, periodic blood counts and liver function are recommended during long-term therapy

CLINICALLY SIGNIFICANT DRUG INTERACTIONS Benzodiazepines produce CNS depres effects when administered with such medications as barbiturates or alcohol

CARCINOGENESIS AND MUTAGENESIS No evidence of carcinogenic potential emerge rats during an 18-month study No studies regarding mutagenesis have been performed.

PREGNANCY Reproductive studies were performed in mice, rats, and 2 strains of rab Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastrosch malformed skull and microphthalmia) were seen in drug-treated rabbits without relationsh dosage Although all these anomalies were not present in the concurrent control group, have been reported to occur randomly in historical controls At 40mg/kg and higher, there evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lo doses Clinical significance of these findings is not known However, increased risk of cong tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam meprobamate) during first trimester of pregnancy has been suggested in several stu Because use of these drugs is rarely a matter of urgency, use of lorazepam during this pe should almost always be avoided Possibility that a woman of child-bearing potential ma pregnant at institution of therapy should be considered Advise patients if they become p nant to communicate with their physician about desirability of discontinuing the drug

In humans, blood levels from umbilical cord blood indicate placental transfer of loraze and its glucuronide

NURSING MOTHERS It is not known if oral lorazepam is excreted in human milk like o benzodiazepines As a general rule, nursing should not be undertaken while on a drug s many drugs are excreted in milk

Adverse Reactions, if they occur, are usually observed at beginning of therapy and gene disappear on continued medication or on decreasing dose In a sample of about 3,500 anx patients most frequent adverse reaction is sedation (15 9%), followed by dizziness (6 weakness (4 2%) and unsteadiness (3 4%) Less frequent are disorientation, depression, sea change in appetite headache, sleep disturbance, agitation, dermatological symptoms, function disturbance, various gastrointestinal symptoms and autonomic manifestations dence of sedation and unsteadiness increased with age Small decreases in blood press have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple age may have been taken Manifestations of overdosage include somnolence, confusion and co Induce vomiting and/or undertake gastric lavage followed by general supportive care, moni ing of vital signs and close observation Hypotension, though unlikely, usually may be contro with Levarterenol Bitartrate Injection U S P Usefulness of dialysis has not been determined

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase do gradually when needed, giving higher evening dose before increasi daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosa may vary from 1 to 10mg/day in divided doses. For elderly or deb tated, initially 1-2mg/day; insomnia due to anxiety or transient situ tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Why one benzodiazepine and not another?

Are you concerned about long-acting metabolites? Many clinicians, as well as pharmacologists, are beginning to draw attention to this problem (see New England Journal of Medicine, April 5, 1979).

In contrast to some older benzodiazepines, Ativan (lorazepam) does not give rise to long-lasting active metabolites. As with all benzodiazepines, you should follow the usual precautions concerning co-administration with other CNS depressants and warn your patients against operating dangerous machinery and motor vehicles.

However, it is noteworthy that Ativan showed no clinical evidence of accumulation even when given in high doses over periods up to 6 months. The half-life of free lorazepam is about 12 hours; steady-state serum levels are attained in 2-3 days. Comparable data for diazepam: 20-50 hours and at least 7-10 days. (The pharmacokinetic profile of a drug can define such characteristics as absorption, distribution, metabolism and elimination but cannot, at present, be directly related to its therapeutic effectiveness.)

Ativan has a convenient b.i.d. or t.i.d. dosage schedule; it is compatible with a long list of other medications and, of course, it is a highly effective anxiolytic agent, as established in numerous nationwide, double-blind, controlled evaluations in thousands of patients.



See important information on preceding page.

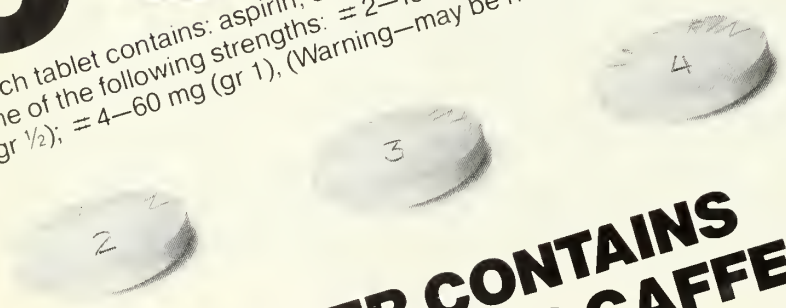
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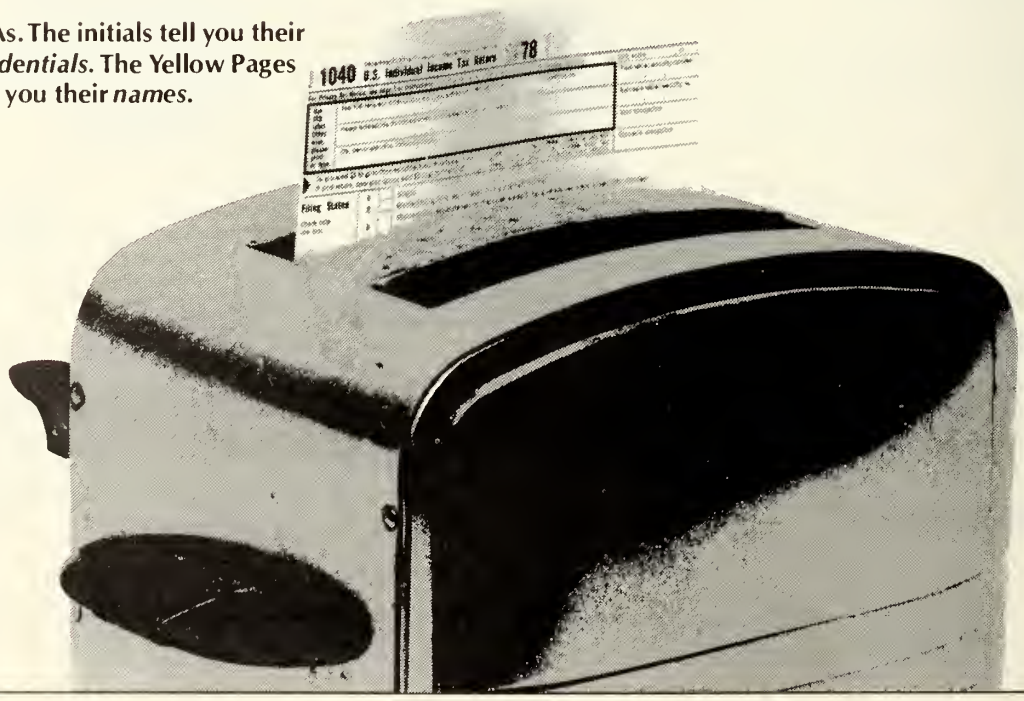
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Council Meeting

Report of Meeting Held January 27, 1980

A meeting of the Council was held on Sunday, January 27, 1980 at the Wichita Hilton Inn beginning at 10:00 AM. Present were Drs. Donald D. Goering, M.D., President, Coldwater; P. J. Antrim, M.D., Anthony; John N. Blank, M.D., Hutchinson; Kenneth Boese, M.D., Manhattan; K. W. Bruner, Jr., M.D., Wichita; Clair C. Conard, M.D., Dodge City; Jack Cooper, M.D., Shawnee Mission; Louis M. Culp, M.D., Kansas City; Ronald R. Davis, M.D., Wichita; Norton Francis, M.D., Wichita; Herbert Fransen, M.D., Newton; Phillip Godwin, M.D., Lawrence; Herman W. Hiesterman, M.D., Quinter; Warren E. Meyer, M.D., Wichita; William J. Reals, M.D., Wichita; Ivan E. Rhodes, M.D., Wichita; Alex Scott, M.D., Junction City; Stephen J. Smith, M.D., Arkansas City; Marvin Snowbarger, M.D., Emporia; Millard C. Spencer, M.D., Topeka; Thomas F. Taylor, M.D., Salina; Max E. Teare, M.D., Garden City; William K. Walker, M.D., Sedan; Roger Warren, M.D., Hanover; Wallace N. Weber, M.D., Hays; and Kermit G. Wedel, M.D., Minneapolis.

Also present was Mrs. Kermit (Kathleen) Wedel, KMS Auxiliary President. Also present were Dwight Allen, Jerry Slaughter, Gary Caruthers, and Val Braun.

The following agenda was considered:

Legislative Update: Hospital cost containment and professional advertising bills were reviewed in detail. Reaffirmed that approval of legislation could be accomplished through the avenues of the Executive Committee, the Council, or the House of Delegates.

Questioned Medicare regulations relating to physicians for billing of services when covered by another physician. Requested a study of this question, including a legal opinion delineating appropriate guidelines, if necessary.

Paging System Dispute — Wyandotte County. Approved that Wyandotte County be requested not to sign an agreement with Mobil Radio Communications. Reaffirmed that KMS could carry the dispute through the appeals process.

Computer. Approved a moratorium on the purchase of an in-house computer at this time, keeping the avenues for future study open.

Nominating Committee Report. Tentative nominees for 1980 are: President: Philip A. Godwin, M.D., Lawrence; President-Elect: Herman W. Hiesterman, M.D., Quinter; First Vice-President: Kermit G. Wedel, M.D., Minneapolis; Second Vice-Presidents: Lew W. Purinton, M.D., Wichita and Roger D. Warren, M.D., Hanover; Constitutional Secretary: Jack R. Cooper, M.D., Prairie Village; Treasurer: William K. Walker, M.D., Sedan; AMA Delegate: Alex Scott, M.D., Junction City; AMA Alternate: Kermit G. Wedel, M.D., Minneapolis; Speaker, KMS House of Delegates: Clair C. Conard, M.D., Dodge City; Vice Speaker, KMS House of Delegates: John O. Yulich, M.D., Kansas City.

Chiropractic. Requested that the Legislative Committee consider drafting appropriate legislation on this topic if necessary.

1980 Dues Billing. Requested staff to distribute a listing of non-AMA members to each county medical society.

Membership Brochure. Approved printing 5,000 copies of the membership brochure. Also authorized preparation and distribution of a Physician Record Guide as part of an overall package of materials relating to all aspects of medical practice.

KaMPAC. Approved contribution of $2,500 to KaMPAC Educational Fund. Requested that nominations to the Congressional Districts 1, 3 and 5 be sent to Dr. Ronald Davis or Gary Caruthers.

Blue Cross Board Appointment. Nominated Thomas F. Taylor, M.D., Salina.

Council District Meetings. Reaffirmed the practice of the KMS President visiting the 19 Council districts at the invitation of the Councilor. Discussed various promotional methods for increasing attendance.

Executive Committee. All previous actions were approved.

Surgical Assistants. Reviewed material pertaining to the reimbursement of Surgical Assistants.

Judicial Committee. A detailed report of committee activities and a proposed budget will be submitted at a later date.

The meeting adjourned at 1:00 PM.

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Indications: For the treatment of mild to moderately severe pneumococcal respiratory tract infections and mild staphylococcal skin and soft-tissue infections that are sensitive to penicillin G. See the package literature for other indications.

Contraindication: Previous hypersensitivity to penicillin.

Warnings: Serious, occasionally fatal, anaphylactoid reactions have been reported. Some patients with penicillin hypersensitivity have had severe reactions to a cephalosporin; inquire about penicillin, cephalosporin, or other allergies before treatment. If an allergic reaction occurs, discontinue the drug and treat with the usual agents (e.g., epinephrine or other pressor amines, antihistamines, or corticosteroids).

Precautions: Use with caution in individuals with histories of significant allergies and/or asthma. Do not rely on oral administration in patients with severe illness, nausea, vomiting, gastric dilatation, cardiospasm, or intestinal hypermotility. Occasional patients will not absorb therapeutic amounts given orally. In streptococcal infections, treat until the organism is eliminated (minimum of ten days). With prolonged use, nonsusceptible organisms, including fungi, may overgrow; treat superinfection appropriately.

Adverse Reactions: Hypersensitivity, including fatal anaphylaxis. Nausea, vomiting, epigastric distress, diarrhea, and black, hairy tongue. Skin eruptions, urticaria, reactions resembling serum sickness (including chills, edema, arthralgia, prostration), laryngeal edema, fever, and eosinophilia. Infrequent hemolytic anemia, leukopenia, thrombocytopenia, neuropathy, and nephropathy, usually with high doses of parenteral penicillin. [102175]

***Equivalent to penicillin V.**

Additional information available to the profession on request.

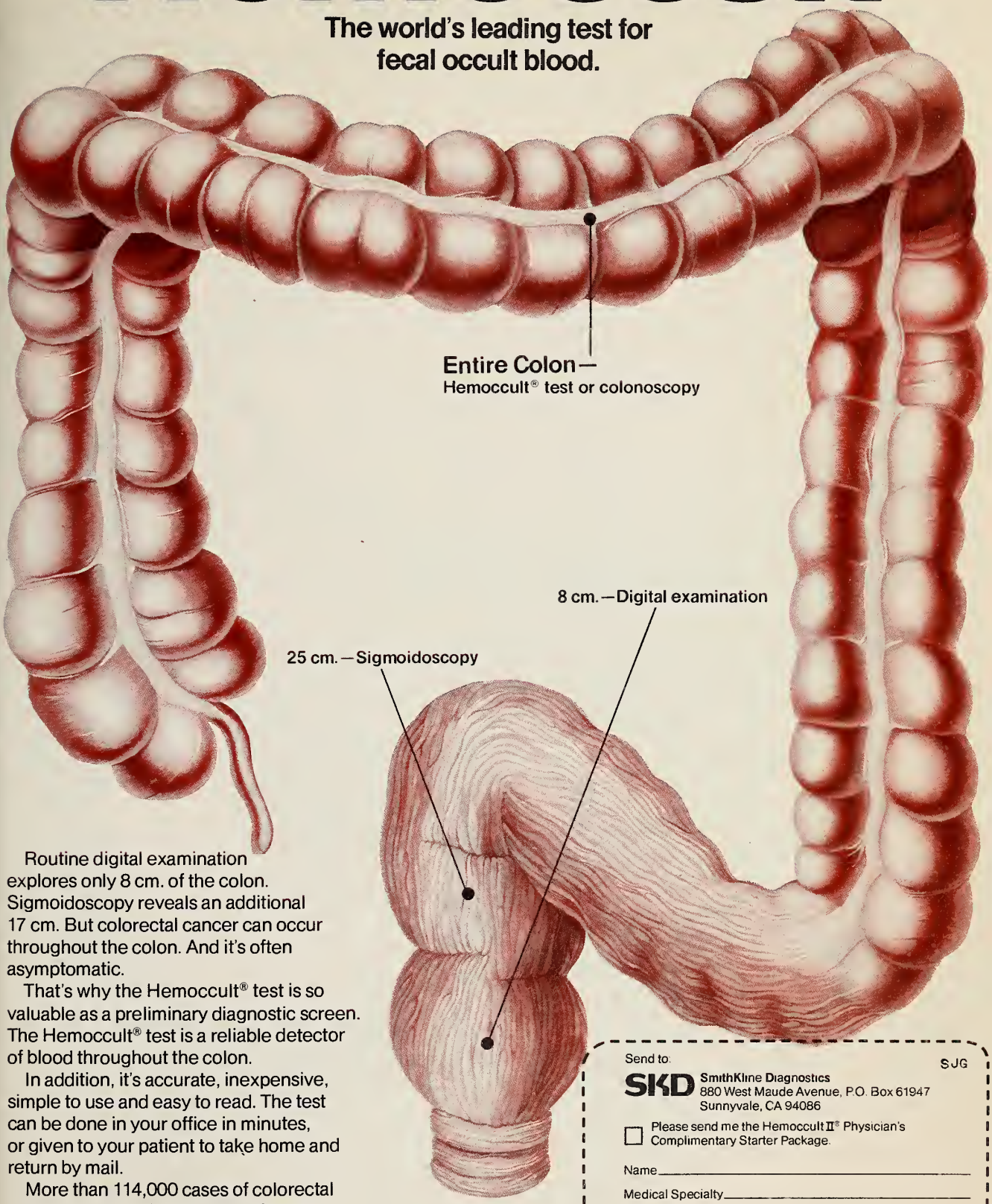
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.in the functional bowel/irritable bowel syndrome*

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(dicyclomine hydrochloride USP)

10 mg. capsules, 20 mg. tablets,
10 mg./5 ml. syrup, 10 mg./ml. injection

helps control abnormal motor activity with minimal anticholinergic side effects†

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In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

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"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

Merrell

8-4420 (Y736A) MNR-804

Bentyl®

(dicyclomine hydrochloride USP)

Capsules, Tablets, Syrup, Injection

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences–National Research Council and/or other information, FDA has classified the following indications as "probably" effective

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis), paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis, myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS: Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness, weakness, dizziness; insomnia, nausea, vomiting, impotence; suppression of lactation; constipation; bloated feeling, severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION: Dosage must be adjusted to individual patient's needs.

Usual Dosage. Bentyl 10 mg. capsule and syrup: *Adults*: 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children*: 1 capsule or teaspoonful syrup three or four times daily. *Infants*: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults*: 1 tablet three or four times daily. Bentyl Injection: *Adults*: 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC. Swiftwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

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Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

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Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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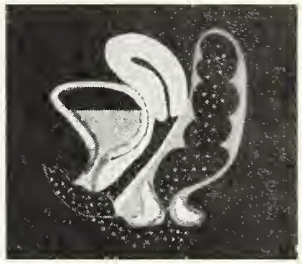
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Bactrim™ DS Double Strength Tablets

Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.



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- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
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- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older.

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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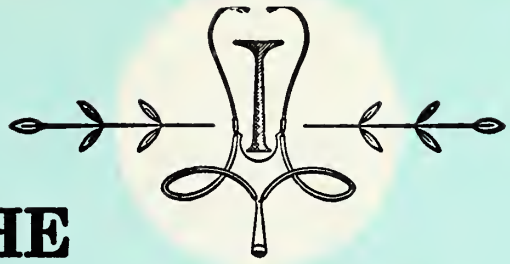
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The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

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Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.



Program Issue

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APRIL
1980

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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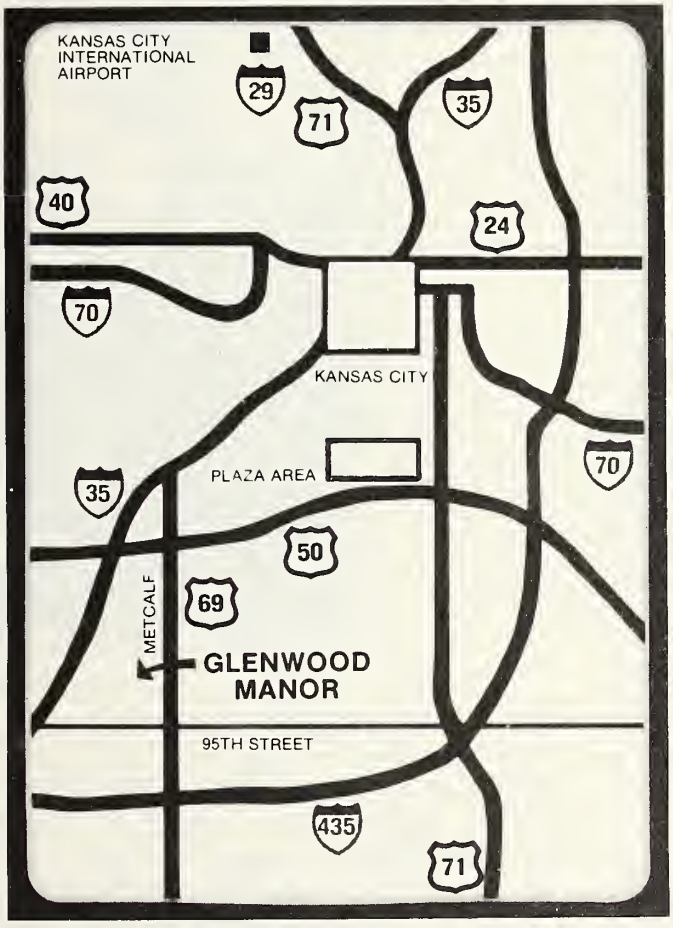
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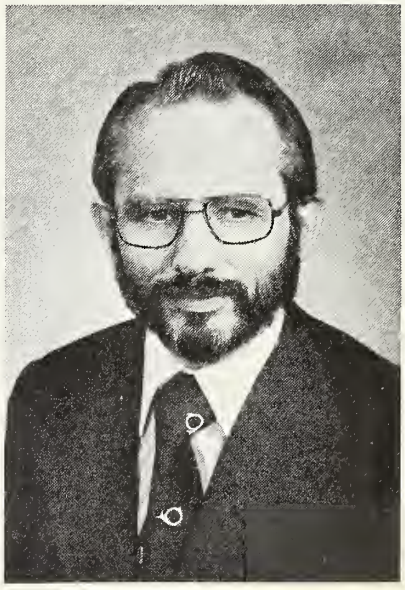
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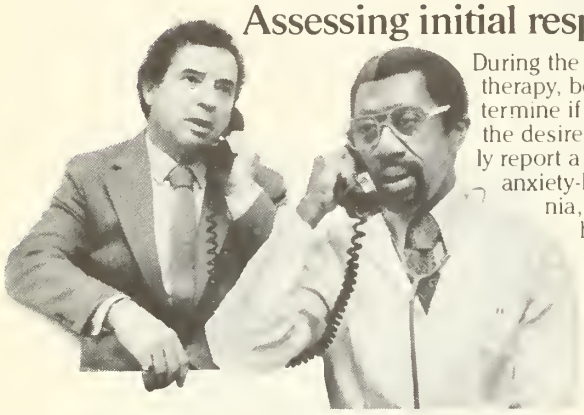
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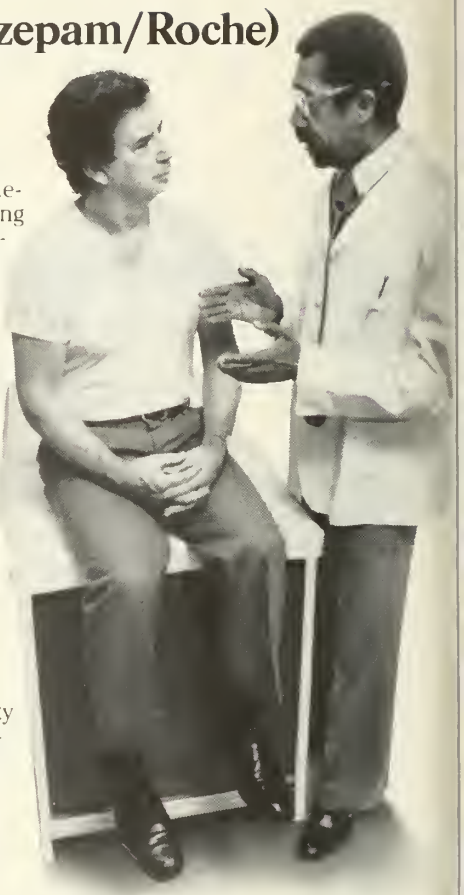
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Monitoring patient response to Valium® (diazepam/Roche)

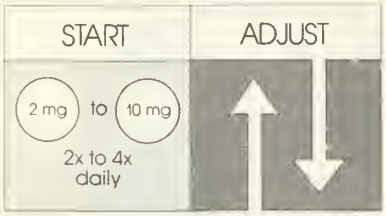


Assessing initial response to therapy

During the first follow-up visit after initiating therapy, both physician and patient should determine if Valium (diazepam/Roche) is having the desired effect. Most patients will promptly report a feeling of relaxation and relief of anxiety-linked symptoms such as insomnia, headaches, palpitations and hyperventilation. You will probably observe that the patient is calmer and more relaxed. If, however, patient response does not measure up to expectations, a reevaluation of the patient's profile with modification of the dosage regimen should be considered.



Making dosage adjustments



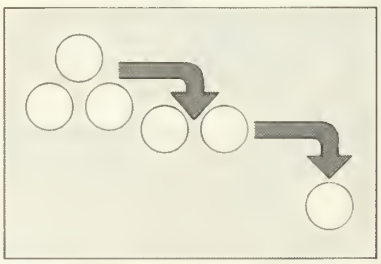
With any psychoactive medication it is good medical practice to initiate therapy at base dosage levels and titrate to the patient's needs. With Valium, experience has shown that 5 mg t.i.d. is usually sufficient although some patients with severe or persistent anxiety may require higher dosages initially. In geriatric or debilitated patients, the recommended dosage is 2 to 2½ mg once or twice daily. When anxiety fluctuates, as is common with most patients, the dosage may be adjusted as needed during the course of therapy; three strengths in scored tablets give you unmatched flexibility and simplicity in individualizing dosage.

Evaluating progress toward therapeutic goals

SET GOALS						
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

At the beginning of therapy it is now common practice for both physician and patient to establish treatment goals and to estimate the amount of time needed to achieve them. Then the patient knows what to expect and when to expect it. Some physicians find that compiling a checklist of presenting symptoms and complaints is useful for assessing the patient's response from visit to visit. In this way, progress toward attainment of the therapeutic goal is reviewed at regular intervals. As patients feel their symptoms abate and begin to develop insight into the sources of their anxiety and psychic tension, the checklist can be expected to dwindle.

Discontinuing pharmacologic intervention



When you decide to discontinue therapy, tapering dosag is good medical practic Although rarely necessary after short-term treatment with Valium, gradual dosage reducti is advisable for patients who have been on extended therapy. This gr ual discontinuance should preclude either recurrence of pretreatment symptoms or development of untoward side effects. Symptoms of withdrawal have almost always been associated with abrupt discontinuance of therapy higher dosages taken continuously over long periods of time.

2-mg, 5-mg, 10-mg scored tablets

Valium® (IV)

diazepam/Roche

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See the following page for a summary of product information.

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Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome, convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

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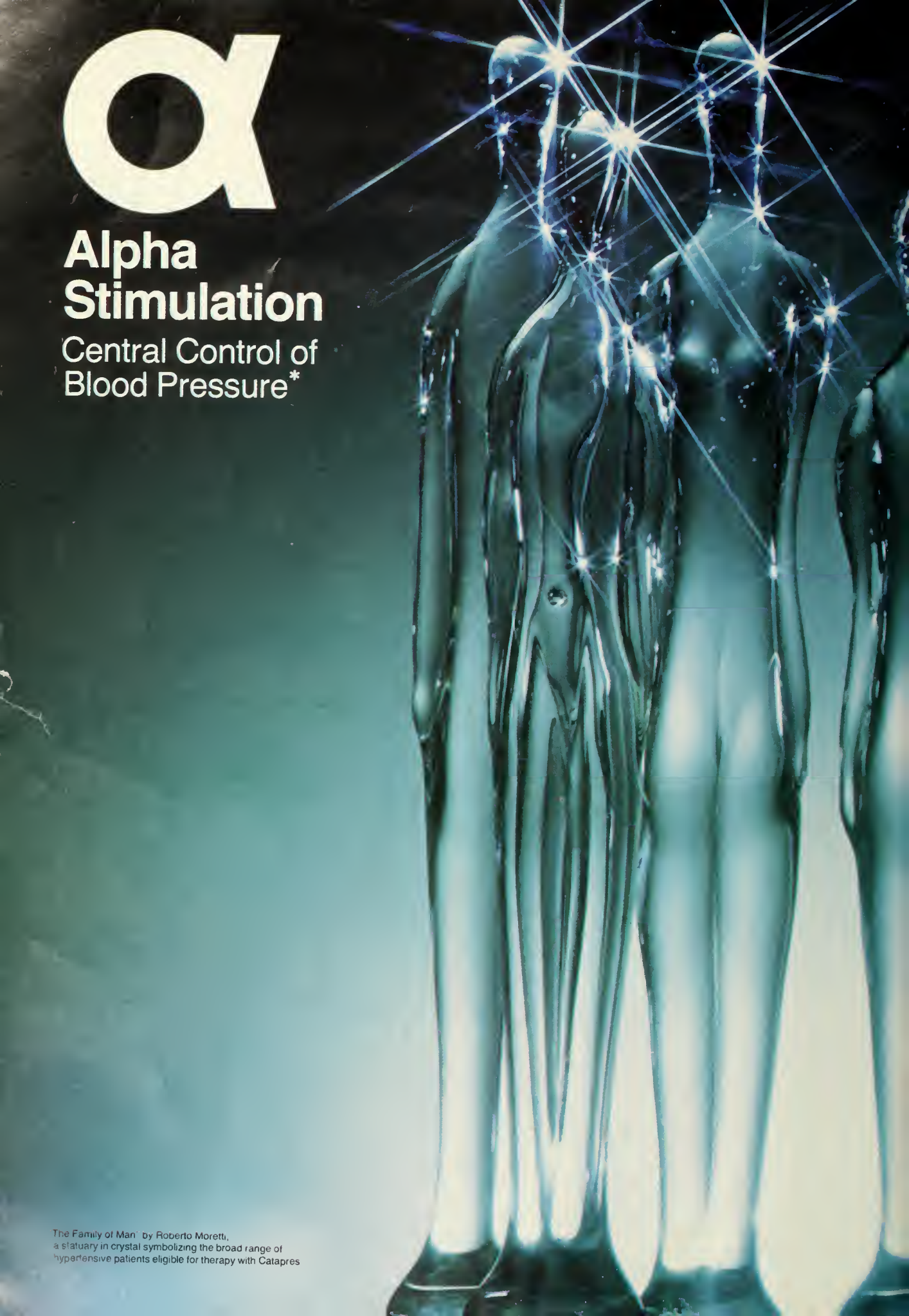
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cimetidine

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and in 8 ml. multiple-dose vials,
both in packages of 10.

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- *Catapres can be useful even in these patients with:*

Congestive heart failure	Allergic rhinitis
Ventricular hypertrophy	Hepatic disease
Hyperglycemia	Hyperuricemia
Diabetes mellitus	Gouty arthritis
Bronchial asthma	Sulfonamide hypersensitivity

Like any antihypertensive, use with caution in severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

work/play—normal hemodynamic responses to exercise maintained.

love—low incidence of impotence and/or loss of libido: 2.8% in 1,923 patients studied.[1]

cardiac output—tends to return to control values during long-term therapy.

blood flow—preserved in kidney.

No Single Advantage Determines Drug Choice.

Other factors must include:

The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

[1] Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

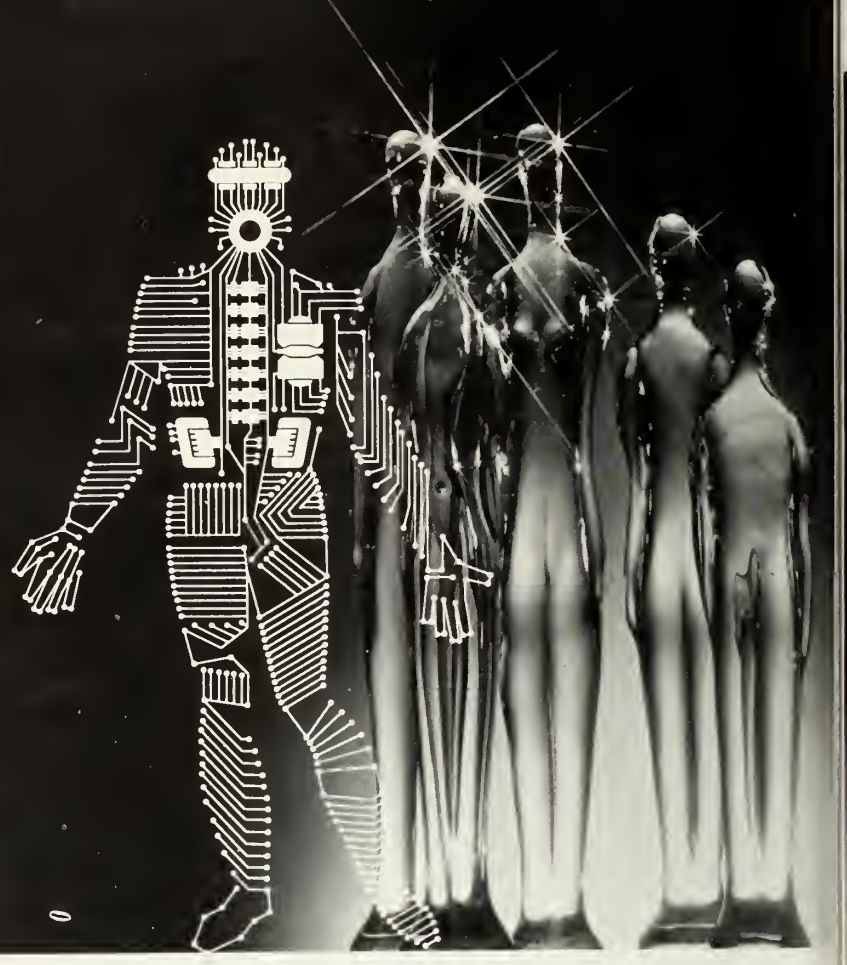
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Hypertension

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Catapres®
(clonidine HCl)

Hypertension

- No contraindications.
- Effective in all degrees of hypertension. It is mild to moderate in potency.
- Low incidence of depression, impotence, orthostatic hypotension—no fatal hepatotoxicity.
- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
(clonidine hydrochloride)
Tablets of 0.1, 0.2, 0.3 mg

Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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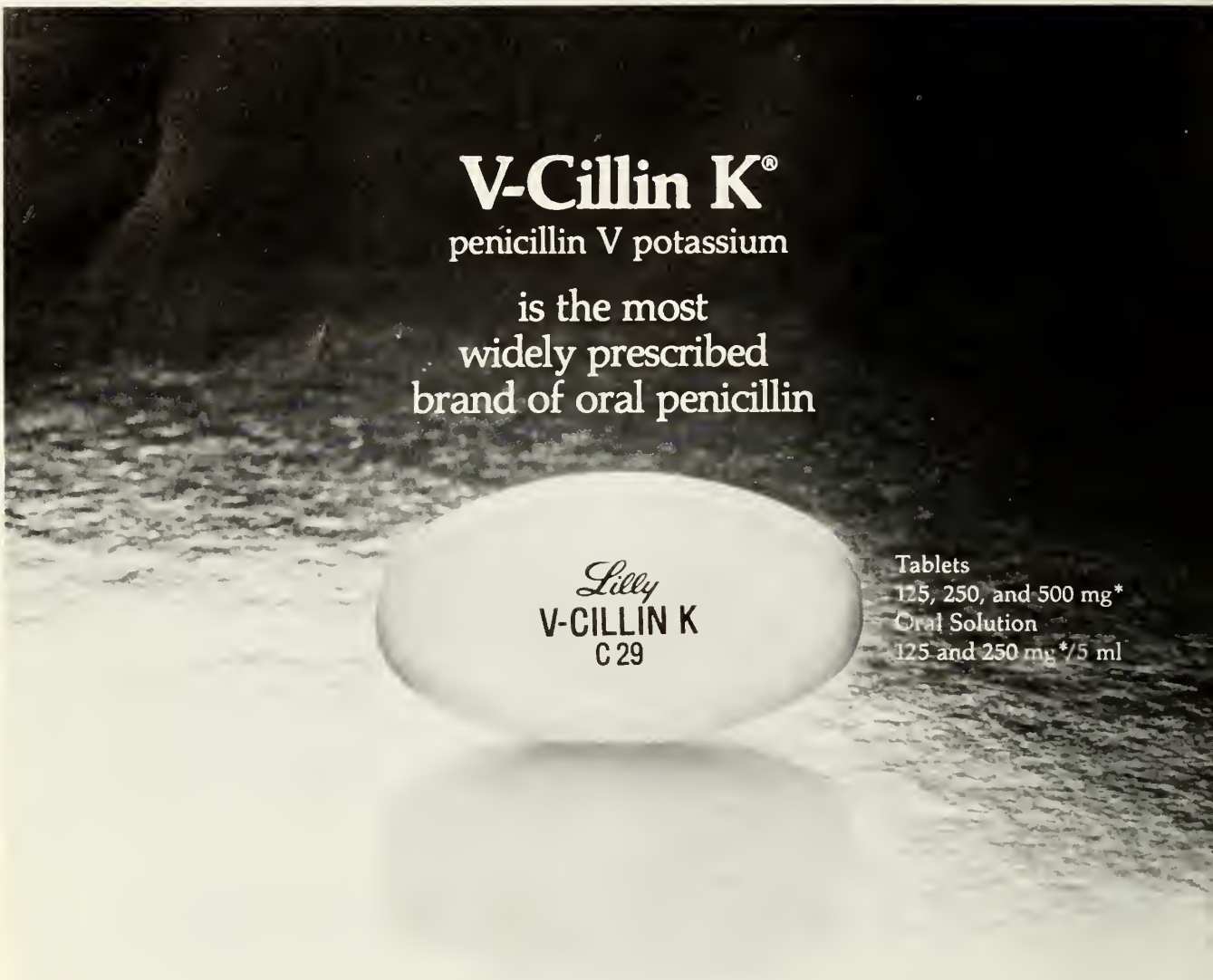
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V-Cillin K*
penicillin V potassium

Description: V-Cillin K is the potassium salt of penicillin V. This chemically improved form combines acid stability with immediate solubility and rapid absorption.

Indications: For the treatment of mild to moderately severe pneumococcal respiratory tract infections and mild staphylococcal skin and soft-tissue infections that are sensitive to penicillin G. See the package literature for other indications.

Contraindication: Previous hypersensitivity to penicillin.

Warnings: Serious, occasionally fatal, anaphylactoid reactions have been reported. Some patients with penicillin hypersensitivity have had severe reactions to a cephalosporin; inquire about penicillin, cephalosporin, or other allergies before treatment. If an allergic reaction occurs, discontinue the drug and treat with the usual agents (e.g., epinephrine or other pressor amines, antihistamines, or corticosteroids).

Precautions: Use with caution in individuals with histories of significant allergies and/or asthma. Do not rely on oral administration in patients with severe illness, nausea, vomiting, gastric dilatation, cardiospasm, or intestinal hypermotility. Occasional patients will not absorb therapeutic amounts given orally. In streptococcal infections, treat until the organism is eliminated (minimum of ten days). With prolonged use, nonsusceptible organisms, including fungi, may overgrow; treat superinfection appropriately.

Adverse Reactions: Hypersensitivity, including fatal anaphylaxis. Nausea, vomiting, epigastric distress, diarrhea, and black, hairy tongue. Skin eruptions, urticaria, reactions resembling serum sickness (including chills, edema, arthralgia, prostration), laryngeal edema, fever, and eosinophilia. Infrequent hemolytic anemia, leukopenia, thrombocytopenia, neuropathy, and nephropathy, usually with high doses of parenteral penicillin. (102175)

***Equivalent to penicillin V.**

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When: May 14 thru 17, 1980

For: Physicians practicing in Kansas

Purpose: To assist the practicing physician in the diagnosis and treatment of rheumatic diseases and to help him fulfill continuing education requirements.

Continuing Education Credit Requested: 20 hrs. Category I

Cost: Registration fee paid by Kansas Chapter, Arthritis Foundation. Physician responsible for meals and lodging.

Sponsored by: Kansas Chapter, Arthritis Foundation, in cooperation with Continuing Education Division, University of Kansas Medical School

RHEUMATOLOGY SYMPOSIUM

"Common Problems in Musculoskeletal Disease"

Time: 8:30 p.m.

Date: Friday, April 18, 1980

Place: Santa Fe Room
Community College
Dodge City, Kansas

Course Credit: Course is approved for 6 hours — Category I, CME credit. As an organization accredited for continuing medical education, the Kansas Chapter, Arthritis Foundation certifies that these continuing medical education offerings meet the criteria for category I credit of the Physician's Recognition Award of the American Medical Association, provided they are completed as designed.

Application made for American Academy of Family Physician CME Credit.

Application made for American Osteopathic Association, 6 hours category I, CME Credit.

Fee: $25.00

FUTURE SYMPOSIUM

"Rheumatic Diseases — 1980 Update"

Date: Friday, November 14, 1980

Place: Wichita, Kansas

Put these dates on your calendar.

For more information write to:

Charles Blackwell, Executive Director
Kansas Chapter, Arthritis Foundation
1602 E. Waterman
Wichita, Kansas 67211

PHYSICIAN — M.D. or D.O.

Kansas Social and Rehabilitation Services, Medical Services Section. Position is vacant for a medical doctor or doctor of osteopathy to administer the Hospital and Medically Related Services Unit. Responsibilities include evaluating services being provided by physicians, hospitals, dentists, and other professionals who are providers in the Medicaid (Medical Assistance) Program recommending policy changes, reviewing services for authorization and assisting the Chief Administrator and staff in other ways in administering the programs. Must be interested in public service and developing a strong recipient oriented program. Salary negotiable. Application and résumé should be submitted to:

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Social and Rehabilitation Services
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SYMPOSIUM:

MODERN THERAPY IN NEUROLOGY

May 19 and 20, 1980

Guest Faculty:

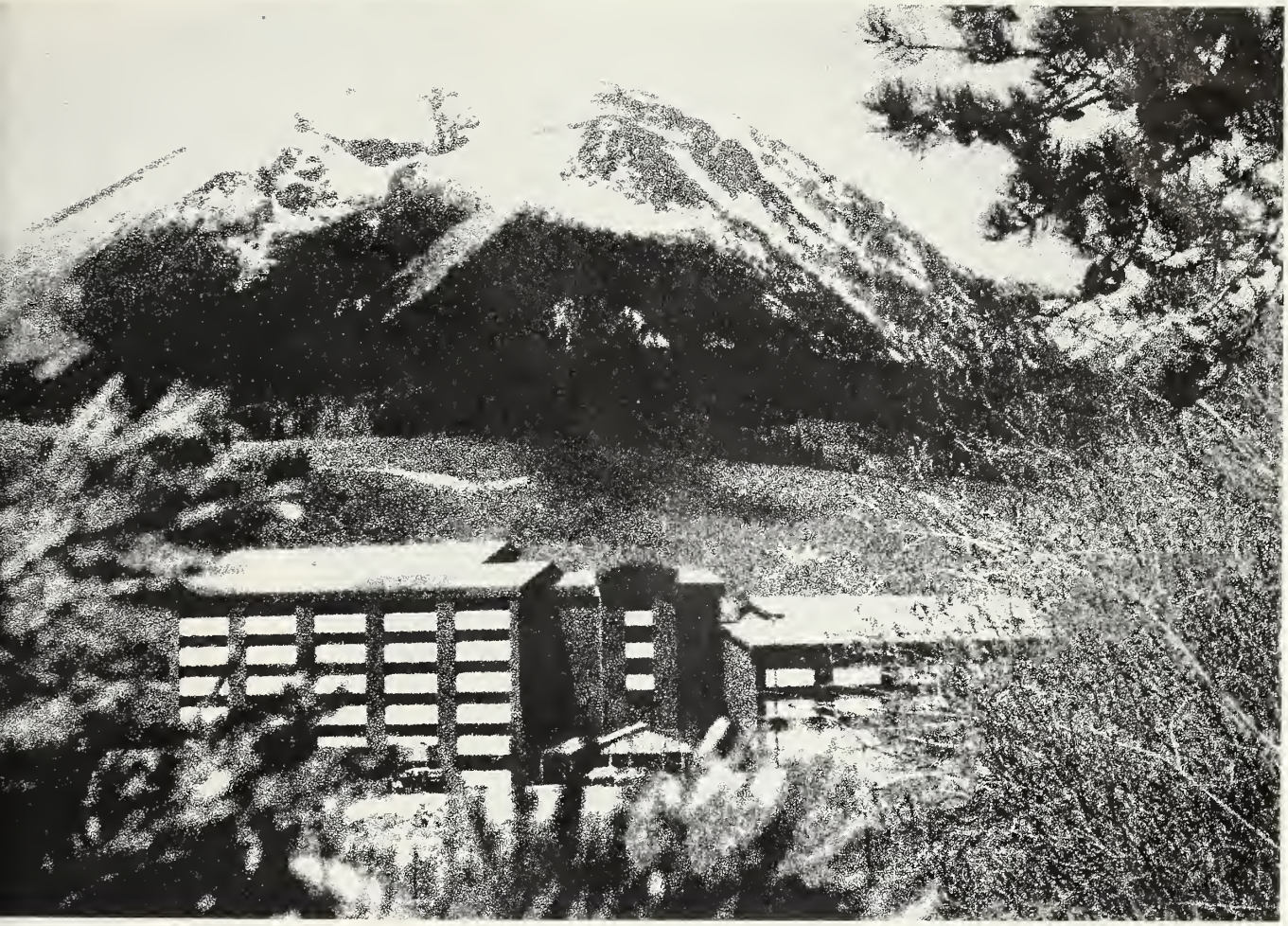
STANLEY FAHN, M.D., Columbia University College of Physicians and Surgeons and Neurological Institute of New York.
GILBERT H. GLASER, M.D., Yale University School of Medicine and Yale-New Haven Hospital, Connecticut.
THEODORE L. MUNSAT, M.D., Tufts New England Medical Center, Boston, Massachusetts.
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Subjects to be discussed will include: PATHOPHYSIOLOGY OF STROKE AND TIA'S; MIGRAINE; CLASSIFICATION OF EPILEPTIC SEIZURES; CHILDHOOD SEIZURES; TREATMENT OF EPILEPSY — RECENT ADVANCES AND CONCEPTS; MOVEMENT DISORDERS — OVERVIEW; NEUROTRANSMITTER RECEPTOR PHARMACOLOGY; MODERN TREATMENT OF PARKINSONISM; HUNTINGTON'S DISEASE; NORMAL STRUCTURE AND FUNCTION OF THE MOTOR UNIT; MODERN APPROACH TO THE PATIENT WITH NEUROMUSCULAR DISEASE; ACHES, CRAMPS AND PAINS.

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Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg, bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg, benzyl benzoate, 12.0 mg, Peruvian balsam, 18.0 mg, zinc oxide, 110.0 mg, also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

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House of Delegates

Glenwood Manor

SATURDAY—MAY 3

7:30 Registration of Delegates
9:00 First Session

SUNDAY—MAY 4

7:30 Registration of Delegates
9:00 Second Session

Council Meeting and Luncheon at Conclusion of House of Delegates

Reference Committee

SATURDAY, MAY 3—2:00 P.M.

Educational Grants

The Kansas Medical Society is grateful for the convention program grants received from:

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Welcome to Kansas City

We welcome all physicians and spouses to Kansas City for the 121st Annual Meeting of the Kansas Medical Society. This is a combined effort of the Johnson County and Wyandotte County medical societies, and we are looking forward to being your hosts during your stay here.

If we can be of any help to you, please do not hesitate to contact us.

John W. Young, M.D., President
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The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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Roy Neil, M.D., Hays (913) 628-3215
George M. Penn, M.D., Topeka (913) 234-9566
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
Virginia L. Tucker, M.D., Lawrence(913) 843-3750
Kermit Wedel, M.D., Minneapolis (913) 392-2144

Kansas Medical Society, Topeka (913) 235-2383/235-3619

PRE-CONVENTION WORKSHOPS

May 1, 1980

Enhancing Your Financial Skills
8:15 AM-4:30 PM
6.5 hours CME Category I — Physicians and Spouses
Financial Planning for the Medical Family
Investments — Insurance — Estate Planning

(To be held at Glenwood Manor — Halls 1 & 2)

Impairment vs Satisfaction
The Physician's Work, Marriage, and Family
1:00-5:00 PM
4 hours CME Category I — Physicians and Spouses

(To be held at Shawnee Mission Medical Center, 74th and Grandview; dinner will follow.)

SPECIALTY SOCIETIES

Friday, May 2

12:00 Kansas Neurosurgical Society
Studio Room 105
Kansas OB/GYN Society
Hall 1 & 2
Luncheon & Business Meeting

Saturday, May 3

12:00 Kansas Society of Anesthesiologists
Luncheon & Business Meeting
Flamingo Room
Kansas Society of Pathologists
Luncheon & Business Meeting
Hall 3
1:00 Kansas Academy of Family Physicians
Board of Directors Meeting
Hall 2
2:00 Kansas Nuclear Medicine Section
Studio Room 105

Sunday, May 4

12:00 Kansas Allergy Society
Luncheon & Business Meeting
Barcelone & Mediterranean



Arden Almquist, M.D.

Speaker for the Sunday Early Bird Breakfast will be Arden Almquist, M.D., Director of Medical Education for Baptist Memorial Hospital, Kansas City, Missouri. Dr. Almquist's topic will be "The Bedside: Where Beliefs Meet."

PRESIDENT'S BANQUET
MAY 3, 1980

Doctor and Mrs. Donald D. Goering extend a personal invitation to members of the Society and their guests to attend the President's Banquet on Saturday evening, May 3, 1980, at 7:00 PM, in the Glenwood Manor Convention Center East.



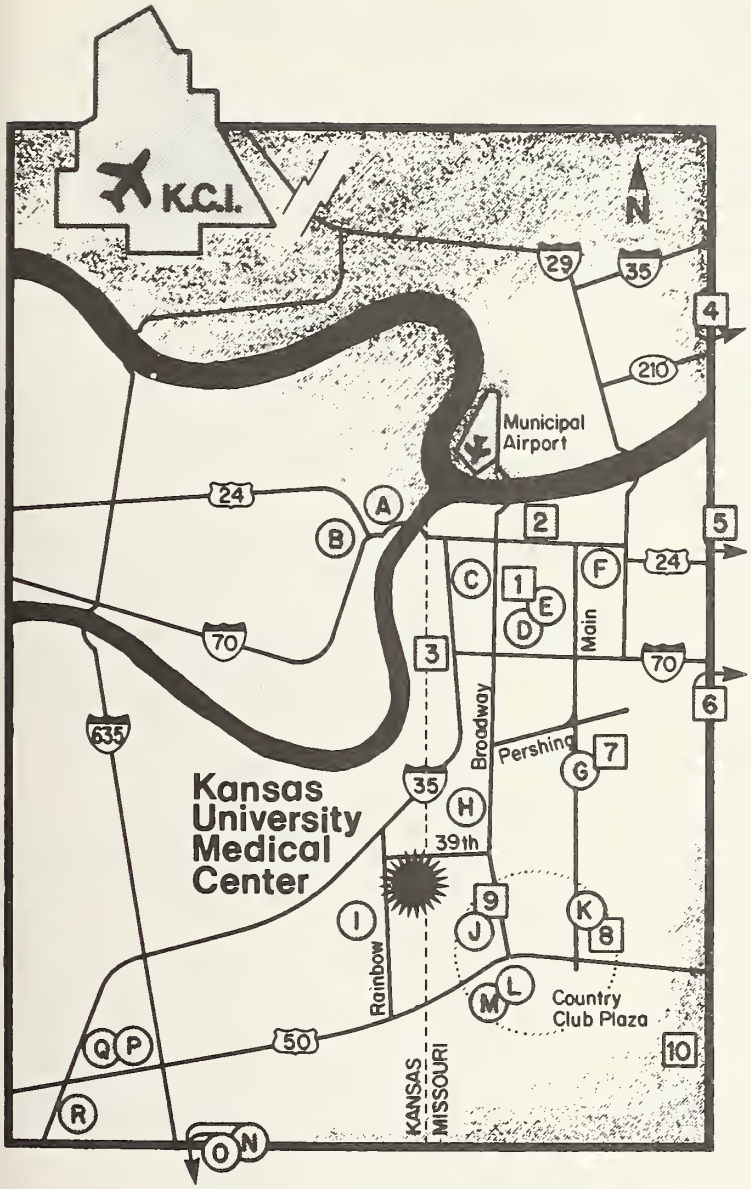
THE K-STATE SINGERS

The K-State Singers combine vocal, instrumental, and dancing talents into a vibrant presentation of jazz, rock, folk, and Broadway show music.

Comprised entirely of non-music majors, the Singers tour Kansas, the nation, and the world as Kansas State University's "singing ambassadors."

They are under the direction of Gerald Polich.

Following the program, the Geri Van Trio will furnish music for dancing.



LEISURE-TIME ACTIVITIES

1. CENTRAL TICKET OFFICE VISITORS INFORMATION
 City Center Square
2. CITY FOOD MARKET
 5th & Delaware
3. KEMPER SPORTS ARENA
 1800 Genesee
4. WORLDS OF FUN
 I-435 & Parvin Rd. Exit
5. TRUMAN LIBRARY
 U.S. 24 at Delaware (Independence, Mo.)
6. HARRY S TRUMAN SPORTS COMPLEX
 I-70 & Blue Ridge Cutoff
7. CROWN CENTER
 24th & Grand
 Shopping & Restaurants
8. NELSON ART GALLERY
 45th & Rockhill Rd.
9. WESTPORT SQUARE
 Westport Rd. at Broadway
 Shopping & Restaurants
10. STARLIGHT THEATRE
 KANSAS CITY ZOO
 63rd & Swope Pkwy.

HOTELS/MOTELS

- (A) HOLIDAY INN
 424 Minnesota
 913-342-6919
- (B) TRAVELODGE
 425 Minnesota
 913-621-3085
- (C) RAMADA INN
 610 Washington
 816-421-1800
- (D) HOLIDAY INN CITY CENTER
 1301 Wyandotte
 816-221-8800
- (E) RADISSON-MUEHLEBACH HOTEL
 12th & Baltimore
 816-471-1400
- (F) SHERATON DOWNTOWN
 6th & Main
 816-842-6090
- (G) CROWN CENTER HOTEL
 1 Pershing Road
 816-474-4400
- (H) TRAVELODGE BROADWAY
 3240 Broadway
 816-531-9250
- (I) BEST WESTERN COLONIAL MOTEL
 3930 Rainbow
 913-236-6880
- (J) GRANADA ROYALE
 220 West 43rd
 816-756-1720
- (K) HILTON PLAZA INN
 1 East 45th Street
 816-753-7400
- (L) HOTEL RAPHAEL
 325 Ward Parkway
 816-756-3800
- (M) ALAMEDA PLAZA
 Wornall Road & Ward Parkway
 816-756-1500
- (N) WHITE HAVEN MOTOR LODGE
 8039 Metcalf
 913-649-8200
- (O) GLENWOOD MANOR
 9200 Metcalf
 913-649-7000
- (P) HOLIDAY INN
 W. 50 Highway & Metcalf
 913-262-3010
- (Q) MISSION INN
 7508 W. 50 Highway
 913-262-9600
- (R) WESTERN HILLS MOTOR HOTEL
 I-35 & 67th Street
 913-432-9400

PARKING

Complimentary parking is available on request in a lot at Olathe Boulevard and Eaton Street just southeast of Battenfeld Auditorium. Check box on **Family Practice** registration card and a priority parking permit will be mailed to you. **If you are enrolling for Infectious Diseases symposium only, please contact Office of Continuing Education for parking permit (Telephone 913-588-4488).**

BUS SERVICE

Charter bus transportation will be available between the Glenwood Manor and Battenfeld Auditorium for members of the Kansas Medical Society. You are requested to check with Kansas Medical Society Meeting Office at Glenwood Manor for bus schedule.

Telephones

Glenwood Manor	913-649-7000
Battenfeld Auditorium	913-588-4488

Sixth Annual Postgraduate Symposium on Family Practice

Battenfeld Auditorium, Student Center-Continuation Study Building
Rainbow Boulevard at Olathe, Kansas City

Presented by: The University of Kansas College of Health Sciences and Hospital, Division of Health Care, Outreach and Continuing Education

In cooperation with: Kansas Division of the American Cancer Society and American Heart Association, Kaw Valley Tri-County Division

As an organization accredited for continuing medical education, The University of Kansas College of Health Sciences designates this continuing medical education activity as meeting the criteria for 11½ credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. Program is acceptable for 11½ prescribed hours by the American Academy of Family Physicians.

Wednesday, April 30

8:00 Coffee
Francisco Lounge

8:10 Registration
Francisco Lounge

8:45 Announcements
Jesse D. Rising, M.D., Professor of Medicine and Co-Director of Continuing Education

8:50 Welcome
Jack D. Walker, M.D., Professor and Chairman, Department of Family Practice
James G. Price, M.D., Associate Professor of Family Practice, Presiding

9:00 The H_2 Blockers (Cimetidine) — An Update
James B. Rhodes, M.D., Professor of Medicine (Gastroenterology) and Physiology

9:30 Evaluation of the Jaundiced Patient
Norton J. Greenberger, M.D., Professor and Chairman, Department of Medicine

10:00 New Concepts in the Management of Viral Hepatitis
James S. Reed, M.D., Assistant Professor of Medicine (Gastroenterology)

10:30 Coffee

11:00 Pancreatitis — An Update
Norton J. Greenberger, M.D.

11:30 The Role of Endoscopy in GI Pathology
James B. Rhodes, M.D.

12:00 Panel Discussion
Drs. Greenberger, Reed, and Rhodes; Dr. Price, Moderator

12:45 Luncheon
Norman G. Marvin, M.D., Assistant Professor of Family Practice, Presiding

2:00 Thyroid Function Tests — Selection and Interpretation
Joseph C. Meek, Jr., M.D., Professor of Medicine (Endocrinology and Metabolism) and Director, Division of Health Care Outreach and Continuing Education

2:30 Diabetic Ketoacidosis — Diagnosis and Management
Joseph L. Kyner, M.D., Associate Professor of Medicine (Endocrinology, Metabolism and Genetics)

3:00 Refreshments

3:30 Osteoporosis — Diagnosis and Management
Barbara P. Lukert, M.D., Associate Professor of Medicine (Endocrinology, Metabolism and Genetics) and Mary J. Roberts Distinguished Chair in Nutrition

4:00 Hypoglycemia — Fact or Fantasy
Joseph L. Kyner, M.D.

4:30 Panel Discussion
Drs. Kyner, Lukert, and Meek; Dr. Marvin, Moderator

5:15 Adjournment

Thursday, May 1

Lawrence L. Perry, Jr., M.D., Assistant Professor of Family Practice, Presiding

9:00 Pediatric Airway Emergencies
John D. Robinson, M.D., Assistant Professor and Director, Residency Program, Department of Anesthesiology; and Co-Director, Pediatric Intensive Care Unit

9:30 Pediatric Renal Diseases
Michael D. Bailie, M.D., Ph.D., Profes-

sor and Chairman, Department of Pediatrics

10:00 Management of the Psychological Aspects of Physical Illness
Paul C. Laybourne, Jr., M.D., Professor of Psychiatry and Family Practice; Associate Professor of Pediatrics; and Director, Division of Child Psychiatry

10:30 Coffee

11:00 Hypertension in Children
Michael D. Bailie, M.D.

11:30 Evaluation of the Sick Scrotum
David R. Stewart, M.D., Associate Professor of Surgery and Pediatrics and Chief, Section of Pediatric Surgery

12:00 Panel Discussion
Drs. Bailie, Laybourne, Robinson and Stewart; Dr. Perry, Moderator

12:45 Luncheon
Sharon Baucom-Copeland, M.D., Assistant Professor of Family Practice, Presiding

2:00 Dr. Bergin's Medicine Show
James J. Bergin, M.D., Professor of Medicine; Director of Medical Education, Bethany Medical Center, Kansas City, Kansas

2:45 Nuclear Medicine — 1980
Ralph G. Robinson, M.D., Associate Professor of Diagnostic Radiology and Director, Division of Nuclear Medicine

3:15 Diagnostic Ultrasound — 1980
S. J. Rosenthal, M.D., Assistant Professor of Diagnostic Radiology

3:45 Radiation Therapy — 1980
Carl M. Mansfield, M.D., Professor and Chairman, Department of Radiation Therapy

4:15 Refreshments followed by Tour of the MID-AMERICA RADIATION CENTER

5:00 Adjournment

Bus Transportation

The following is a tentative schedule of bus service for the Infectious Disease Symposium (one 45-passenger bus):

7:45 First trip to leave Glenwood Manor for KU Battenfeld Auditorium (Discharge passengers and immediately return to Glenwood Manor)
8:30 Second trip to leave Glenwood Manor for KU Battenfeld Auditorium
12:30 Leave Battenfeld Auditorium for Glenwood Manor
1:15 Leave Glenwood Manor for Battenfeld Auditorium
5:00 Leave Battenfeld Auditorium for Glenwood Manor
5:00 (Other Buses) Leave Battenfeld Auditorium for Alameda Plaza
6:15 Leave Alameda Plaza for Glenwood Manor

Twenty-Fourth Annual Symposium on Infectious Diseases

Battenfeld Auditorium, Student Center-Continuation Study Building
Rainbow Boulevard at Olathe, Kansas City

Presented by: American Academy of Family Physicians; The University of Kansas College of Health Sciences and Hospital, Division of Health Care Outreach and Continuing Education; and the Kansas Medical Society.

As an organization accredited for continuing medical education, The American Academy of Family Physicians designates this continuing medical education activity as meeting the criteria for 6 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. Program is acceptable for 6 prescribed hours by the American Academy of Family Physicians. Credit will be issued by the American Academy of Family Physicians.

Friday, May 2

8:00 Coffee
Francisco Lounge

8:10 Registration
Francisco Lounge

8:55 GREETINGS
Jesse D. Rising, M.D., Professor of Medicine and Co-Director of Continuing Education, UKSM

9:00 WELCOME
John S. Derryberry, M.D., President, The American Academy of Family Physicians, Shelbyville, Tennessee

9:05 INTRODUCTION
Chien Liu, M.D., Professor of Medicine and Pediatrics; Director, Division of Infectious Diseases; and Program Coordinator of Infectious Diseases Symposium
Loren H. Amundson, M.D., Chairman, American Academy of Family Physicians Committee on Continuing Medical Education, Sioux Falls, South Dakota, Presiding

9:10 MANAGEMENT OF ACUTE RESPIRATORY INFECTION IN PATIENTS WITH CHRONIC LUNG DISEASE
James E. Fish, M.D., Assistant Professor of Medicine, Respiratory Division, Johns Hopkins University School of Medicine, Baltimore, Maryland

9:35 DIAGNOSIS AND MANAGEMENT OF ACUTE URINARY TRACT INFECTIONS
Terence C. Davies, M.D., Professor and Chairman, Department of Family Practice, University of Michigan Medical School, Ann Arbor, Michigan

10:00 Coffee

10:20 CURRENT STATUS OF ANTIBIOTICS — 1980
Robert C. Moellering, Jr., M.D., Associate Professor of Medicine, Harvard Medical School; and Associate Physician, Infectious Disease Unit, Massachusetts General Hospital, Boston, Massachusetts

10:45 PEDIATRIC UPPER RESPIRATORY INFECTIONS
Adnan S. Dajani, M.D., Professor of Pediatrics and Director, Division of Infectious Diseases, Wayne State University School of Medicine and Children's Hospital, Detroit, Michigan

11:10 QUESTIONS AND PANEL DISCUSSION
Drs. Dajani, Davies, Fish, Liu, and Moellering; Robert W. Weber, M.D., Clinical Associate Professor of Medicine, UKSM, Moderator

12:30 Luncheon
Richard R. Brummett, M.D., Member, American Academy of Family Physicians Commission on Education, Salina, Presiding

2:00 INFLUENZA
James E. Fish, M.D.

2:25 DIAGNOSIS AND MANAGEMENT OF CHRONIC URINARY TRACT INFECTIONS
Terence C. Davies, M.D.

2:50 Coffee

3:10 ANAEROBIC INFECTIONS
Robert C. Moellinger, Jr., M.D.

4:00 QUESTIONS AND PANEL DISCUSSION
Drs. Dajani, Davies, Fish, Liu, and Moellering; Dr. Weber, Moderator

5:00 KAMPAC HOSPITALITY SUITE, Crystal Room
Cocktails and Hors d'Oeuvres
Everyone invited

7:00 AMAERF AUCTION, Patio
Sponsored by the Kansas Medical Auxiliary

Bus transportation will be available between Glenwood Manor and Battenfeld Auditorium

Saturday, May 3

7:00 Kansas Foundation for Medical Care
Hall No. 4
7:30 Registration
Convention Center Lobby
7:30 Past Presidents Breakfast
Emerald Room
9:00 First House of Delegates
Convention Center West
12:00 Delegates Luncheon
Crystal Room
2:00 Reference Committee
Convention Center West
5:30 Reception for Physicians and Spouses
Crystal Room
Sponsored by KU Alumni Association
7:00 Annual President's Banquet
Convention Center East
Entertainment — K-State Singers
Dance — Geri Van Trio

Sunday, May 4

7:30 Registration
Convention Center Lobby
7:30 Early Bird Breakfast
Crystal Room
"The Bedside: Where Beliefs Meet"
Arden Almquist, M.D., Baptist Memorial Hospital, Kansas City, Missouri
9:00 Second House of Delegates
Convention Center West
12:30 Council Luncheon and Meeting
Crystal Room

SPORTS ACTIVITIES

Friday, May 2
Glenwood Manor &
Wolf Creek Country Club

Arrangements may be made at the registration desk.

SPORTS DINNER — Friday night

GENERAL INFORMATION

Wednesday, April 30

KU FAMILY PRACTICE SYMPOSIUM
Battenfeld Auditorium

Thursday, May 1

KU FAMILY PRACTICE SYMPOSIUM
Battenfeld Auditorium

7:30 Registration
Glenwood Manor Convention Center
8:15 Enhancing Your Financial Skills
Halls 1 & 2
1:00 The Physician's Work, Marriage, and Family
Shawnee Mission Medical Center

Friday, May 2

7:30 Registration
Convention Center Lobby
8:30 Infectious Diseases Symposium
Battenfeld Auditorium
10:00 Golf Activities
Wolf Creek Country Club
5:00 KaMPAC Hospitality Suite
Crystal Room
7:00 AMAERF Auction
Patio

Saturday, May 3

7:30 Registration
Convention Center Lobby
7:30 Past Presidents Breakfast
Emerald Room
9:00 First House of Delegates
Convention Center West
12:00 Delegates Luncheon
Crystal Room
2:00 Reference Committee
Convention Center West
5:30 Reception for Physicians and Spouses
Crystal Room
(KU Alumni Association)
7:00 Annual President's banquet
Convention Center East

Sunday, May 4

7:30 Registration
Convention Center Lobby
7:30 Early Bird Breakfast
Crystal Room
9:00 Second House of Delegates
Convention Center West
12:00 Council Luncheon and Meeting
Crystal Room

Councilor Reports

Activities in the Council Districts of Kansas

DISTRICT 1

During the past year, Council District 1 has continued to approach national norms in terms of net physician numbers and doctor:patient ratios. Although some physician recruiting may be characterized as economically — rather than medically — motivated, the District 1 situation is satisfactory.

Our meeting with KMS President Donald D. Goering was pleasant and informative. These meetings provide another open channel of communication between the KMS office and our membership, and are well received.

It is axiomatic that government financed health care will focus on costs and control. Medicare and Medicaid experiences have demonstrated that physician and patient are subjected to both. However, it appears that a growing number of Kansas patients have adopted a position athwart liberal efforts toward national health insurance. In this context, our patients are able to appreciate reasonable, informative opinions about the unhappy interface between politics and medicine.

We are the best informed group in the health care discourse. This is not a time for aloof silence. Our present contribution to this topic can influence the freedom that we and our patients must have. The best medical care system in the world is at stake.

ROGER D. WARREN, M.D., *Councilor*

DISTRICT 2

Council District 2, which consists of the Wyandotte County Medical Society, has kept busy with the routine types of things that county medical societies handle. We have continued our activity in the emergency medical care field, and our Emergency Medical Care Committee is the hardest working and most active committee in the Society.

One other area of activity for the Society this year was to assist our City-County Health Department in their efforts to maintain a budget that would continue to provide all of the services we have come to expect from our Health Department. The city had prepared a budget that would have drastically curtailed the services of the health department. Representatives of the Society testified at public hearings in support of maintaining an adequate budget for the health department. Their budget was restored by the city with the use of federal revenue sharing monies. The Society's representatives also met with Senator Jan Meyers to discuss ways of making more state money available to local health departments. The state provides less than one per cent of the funding necessary for our health department, yet they have many programs, rules, and regulations that the local health departments must carry out.

For the first time in many years, our Society lost more members during the year than it gained, resulting in a net decrease of 10 members. This is disturbing when one considers that we added 33 new members during the year. One of the projects for 1980 is the appointment of a Goals Committee to take a good, hard look at what the county society is doing and what it should be doing. We hope to revitalize our Society with the help of this committee.

LOUIS M. CULP, M.D., *Councilor*

DISTRICT 3

The Johnson County Medical Society increased its membership by 20 active members and nine corresponding members during the year. Our membership on December 31, 1979, consisted of 226 active, 77 corresponding, 3 honorary, 4 retired, and 2 leave-of-absence members.

In May, the members of the Johnson County Medical Society were guests of the Johnson County Bar Association at a dinner meeting. The speaker, Kansas City attorney Larry L. McMullen, talked on malpractice screening panels. A committee of attorneys and physicians from Johnson County has been working together on a policy for malpractice screening panels in our county.

In October, the Area Medical Council, a group representing the medical societies in this locale, sponsored "An Evening With Annis." More than 600 people attended and enjoyed a delicious meal at the Alameda Plaza, followed by Dr. Edward Annis' speech. Dr. James J. Muehlberger, Johnson County Medical Society, was chairman of the affair. It was so well received that the AMC is considering another such event for 1980.

The Johnson County and the Wyandotte County Medical Societies will co-host the KMS Annual Meeting at Glenwood Manor, Overland Park, Kansas, on May 2-4, 1980.

LEWIS G. ALLEN, M.D., *Councilor*

DISTRICT 4

District 4 welcomed KMS President, Don D. Goering, M.D., to its annual meeting Tuesday, January 29, 1980, at Crestwood Country Club, Pittsburg. Dr. Goering addressed legislation of concern to medicine being considered in the current legislative session. Of particular interest to members was the KMS sponsored professional advertising bill. Members present expressed approval of this legislation. Also discussed was legislative activity regarding definition, licensing, and regulation of non-physician health providers. Dr. Goering also provided information on the KMS impaired physician program.

RODNEY K. ODGERS, M.D., *Councilor*

DISTRICT 5

The annual meeting of the Council District 5 was held in October 1979, with President Donald D. Goering, M.D., attending. Also present was Jerry Slaughter, Executive Director, from the Executive Office in Topeka.

Unfortunately, the Councilor was out of town, so all he can report is hearsay. There was no other activity involving the Council during the year.

KENNETH M. BOESE, M.D., *Councilor*

DISTRICT 6

During the past year we have again been blessed with good leadership and a sense of cooperation in our county medical society. Dan Kelly served as an excellent President and Chairman of the Board of Directors, then passed the gavel to John (Jack) Runnels, who will also be a hard act to follow.

Byron Cook, our new and outstanding Executive Secretary, is being assisted by a very clever and attractive young lady, Pam Powell. Together they are producing a turnaround in the organizational pattern of the Shawnee County Medical Society. Our Society office now has regular hours, new equipment, and additional services — including Notary Public services, a mailing label service, and an up-to-date and effective membership roster. The Physician Referral Service has been made more systematic and reorganized in an attempt to supply more current and accurate referrals.

The By-laws were completely revised for the first time since 1970. The membership accepted the changes without alarm. *The Bulletin,* which had been inactive since 1976, has been reactivated and is an excellent publication. It has allowed us to maintain more efficient communication with members regarding projects of both Shawnee County Medical Society and Shawnee County Medical Foundation. During the past year, 22 new active members have been added to the roster. Their personal data and pictures have all been published in the SCMS *Bulletin.*

An HMO Committee was formed during the first part of 1979, to objectively examine the HMO issue in Topeka and to educate the Society. Late in the year, the scope of the committee's purpose was expanded to study alternate health care delivery systems. Hall Harrison led this committee through its initial year.

Dan Kelly agreed to serve on the board of the Community Health Care Alternatives in an effort to maintain some input from the Society and to keep the Society and general committee informed about CHCA. Recently, Dan has felt it necessary to resign as a member of this board. He believes that his presence has been utilized by the board to add prestige to their membership and to add credence to their application to the Department of HEW for funding of an HMO. The CHCA Committee is currently working under a $75,000 feasibility study grant and would seem to be committed to the formation of an HMO, even if their investigation does not show cause to search for this form of alternate health care in our community.

With the ever expanding role of medical education in the community, and with a desire to reunite efforts from the Society and from the Shawnee County Medical Foundation, the Society recently formed a Building Committee. We are hopeful that we may come forward soon with a positive and utilizable solution to our housing problems. It would be the desire of the members to develop a facility centrally located in the medical community, and hopefully in close proximity to the Health Sciences Library.

During the past year, the Shawnee County Medical Foundation has continued to serve as the primary guiding force for the graduate and postgraduate medical education in our community. Throughout this period, 241 of our members have received a total

of 5,805 Category I CME credit hours. We have continued our residency program relationship with the University, and have enjoyed having residents in pediatrics, general surgery, internal medicine, and obstetrics and gynecology from the University program. A total of 43 University residents have rotated through our local programs. Of these 43, 29 have been from the Department of Internal Medicine. We have also worked closely with the Menninger Foundation and have supplied appropriate training experiences for eight of their residents during their categorical year.

Two of our community hospitals have completed building programs and the third is working toward its date of completion. Fortunately, we are still not overbedded in our medical community, and continue to have a good profile of health services utilization in Shawnee County. The rather intense competitive relationship between the hospitals seems to be somewhat defused, and we are all looking forward to a more cooperative relationship in the community.

What will 1980 bring to Shawnee County? Very likely we will find ourselves involved with an HMO. We will find it necessary in our hospitals to reevaluate our membership and credentialing process in relationship to limited practitioners in the community. Undoubtedly we will require more of our members' time addressing the legislature and legislative groups, as it becomes ever more apparent that our service patterns will be affected as greatly by legislative act as by medical and technological advance. A Public Relations Committee, such as ours under the leadership of Jim Ransom, will need to become more active and coordinate efforts between our local societies, our specialty societies, and the AMA.

Join and work in your local specialty society, join and contribute to the AMA and its vital political programs. Our continued united front is necessary. We cannot allow our own programs to arrive at the same confused and illogical state currently tolerated in our federal and state governing systems.

MILLARD C. SPENCER, M.D., *Councilor*

DISTRICT 7

Council District 7 includes Lyon, Morris, Waubaunsee, Osage, Coffee, and Chase counties. Approximately 43 of 46 active MDs in this district are members of the Flint Hills Medical Society, of whom 37 are members of the staffs of Newman Memorial Hospital and St. Mary's Hospital in Emporia. Both of these hospitals are completing multi-million dollar expansion programs with the addition of much new and sophisticated equipment and facilities.

We are saddened to have lost from our ranks by death Drs. Edward J. Ryan and Richard F. Conard. However, several new MDs plan to begin practice here soon. These include John Best, M.D., and Bobby Ellis, M.D., internal medicine; Raman Chopra, M.D., pediatrician; Douglas Amend, M.D., obstetrician-gynecologist; and possibly a full time psychiatrist. Collyer Kelling, M.D., began practice of radiology here within the past year. Some 13 or 14 physicians employed by the Physicians Placement Group, Inc., singly provide adjunctive emergency room care at Newman Memorial County Hospital on weekends and holidays.

The Flint Hills District Medical Society, under the presidency of James Glenn, M.D., in 1979 and Stephen Knecht, M.D., in 1980, has conducted monthly meetings (except during the summer) mostly at the now newly remodeled Emporia Country Club of which J. P. Brockhouse, M.D., is president. Most months we have enjoyed good presentations from scientific speakers, usually from out of town. In November 1979 we had a good, informative, political meeting with President Donald Goering, M.D., Executive Director Jerry Slaughter, and Executive Assistant Gary Caruthers. Other Flint Hills Medical Society officers this year are Scott Ryan, M.D., Vice President; Dave Edwards, M.D., Secretary-Treasurer; and Don Coldsmith, M.D., Program Chairman.

Medical services in this district are considered excellent, and few complaints regarding physicians have been brought to our attention.

MARVIN D. SNOWBARGER, M.D., *Councilor*

DISTRICT 8

Only minor incidents occurred with a measure of excitement for Cowley County and District 8. New members are Drs. Frank Collins, Ashok Shah, and Joseph Leiker. Retired physicians are Drs. James Hill and Winthrop Hoyle.

The number one goal for 1980 is opposition to all national health insurance and socialistic propaganda, including AMA intervention. Any physician interested in retaining freedom of choice and the private practice of medicine should join the Association of American Physicians and Surgeons. If interested, write to me at 115 Radio Lane, Arkansas City, KS 67005.

STEPHEN J. SMITH, M.D., *Councilor*

DISTRICT 9

Council District 9 has been quiet during the past year. There have been some minor changes in medical personnel, but overall the area is stable. There are additional medical programs being undertaken in Salina, and additional physicians have started practice in the area. Donald D. Goering, M.D., President of the Kansas Medical Society, spoke at a meeting in January 1980 in Salina, and his words of wisdom were well received.

H. D. Doubek, M.D., *Councilor*

DISTRICT 10

The physicians and their spouses of Council District 10 met at the Prairie Dunes Country Club, Hutchinson, on February 27, 1979. W. E. Meyer, M.D., and Gary Caruthers spoke to the group.

The physicians of McPherson, Marion, and Harvey Counties have each hosted the Tri-county Medical Society meetings in their area during the past year.

District 10 physicians will meet with President Donald D. Goering, M.D., at Halstead on April 22, 1980.

Herbert Fransen, M.D., *Councilor*

DISTRICT 11

January 1979:

John J. Schleuter, M.D., accepted the gavel of president of the Medical Society of Sedgwick County. One of his most important goals was implementation of the recommendations of the 1978 MSSC Cost Containment Commission.

Robert K. Purves, M.D., died on January 6, 1979. He had served 25 years as a member of the KMS House of Delegates, and for three years as president of Kansas Blue Shield.

The MSSC-HSA Radiology Ad Hoc Committee submitted a policy statement on radiation therapy services to the planning Committee of the Health Systems Agency of Southeast Kansas, Inc., noting that megavoltage therapy services are available in six metropolitan centers within or just outside the HSASEK area.

February 1979:

Panelists representing local pharmacists and Kansas Pharmacists' Association, chaired by William C. Swisher, M.D., Secretary of the Kansas Healing Arts Board, discussed Kansas brand exchange law at the general membership meeting.

MSSC Legislative Committee, chaired by K. William Bruner, Jr., M.D., was very active during the session of the Kansas Legislature, meeting weekly and coordinating our efforts with KMS Legislative Committee. The annual dinner meeting with the legislators of Sedgwick County and spouses was hosted by MSSC.

A 15-minute live program was aired every two weeks on KAKE-TV, featuring local physicians. This was developed by the Public Relations Commission.

March 1979:

AMA Washington lobbyist Ken Benjamin was the featured speaker at the March general membership meeting.

John E. Schlicher, M.D., replaced Hugo Weber, M.D., as physician representative to the Board of Directors of HSA of Southeast Kansas, Inc. Dr. Weber has served medicine in this capacity commendably for one and one-half years.

MSSC membership was surveyed and the Public Relations Commission recommendation for the society's physician news media code was adopted.

The Speakers' Bureau was also reorganized with 70 physicians agreeing to participate.

April 1979:

Derek Shafer, chief attorney for the Kansas Insurance Department, was the featured speaker this month, discussing effects of Kansas malpractice legislation passed two years ago.

Our Auxiliary contributed $2,977.50 to the MSSC Medical Careers Loan Fund. Since 1961, 30 medical students and seven paramedical students have received loans totaling $96,948.00.

May 1979:

Ivan E. Rhodes, M.D., member of the KMS Committee for the Impaired Physician, and William C. Swisher, M.D., Secretary of the Healing Arts Board, discussed the KMS Program for the Impaired Physician.

Zane R. Boyd, M.D., was named president-elect of MSSC for 1980.

MSSC was honored to have Warren E. Meyer, M.D., a past president of MSSC, serve as President of Kansas Medical Society for 1978-79, and in appreciation presented him with a watch at the KMS President's Banquet.

Katherine Pennington, M.D., was honored as one

of five "Women of Achievement" in the community by the Wichita Professional Chapter of Women in Communications.

June 1979:

The Boeing Company requested MSSC to develop an in-hospital utilization review program for its employees and their dependents. The Board requested MSSC Foundation for Medical Care, Inc., President, Ivan E. Rhodes, M.D., to develop such a program for The Boeing Company, and other industrial groups, as a mechanism for providing medical care in a cost effective manner. The project is expected to be implemented in 1980.

MSSC terminated its utilization review program for Medicaid Intermediate Care nursing facilities, but will continue utilization review for all skilled nursing services under Medicare and Medicaid in Sedgwick County.

July 1979:

The general membership of MSSC, at a special meeting, approved the concept for the proposed utilization review program for The Boeing Company employees and their dependents.

August 1979:

A press conference was held to inform the public of the MSSC Medical Care Review Program. Beech Aircraft Company and Kansas Blue Cross-Blue Shield requested an opportunity to participate in this review program in Sedgwick County.

K. William Bruner, M.D., appeared before the Legislative Interim Committee and testified concerning credentialing of health care providers.

Ivan E. Rhodes, M.D., testified before the Legislative Cost Containment Committee opposing proposed hospital rate review legislation.

September 1979:

Annual Sports Day and Recognition Awards Banquet was held. MSSC President John J. Schleuter, M.D., was the winner of the E. S. Edgerton Traveling Golf Trophy. Drs. Wayne Bartlett, Charles Hinshaw, and Clyde Miller were honored for service to their profession and community.

A Medical Advisory Committee to the local health department was established to improve relationships between the health department and the medical community.

The mixed medical bowling league begins its 20th year.

October 1979:

Donald L. Cooper, M.D., team physician for Oklahoma State University, was the featured speaker on "Early Care of Athletic Injuries."

MSSC sponsored a one-day cost containment seminar and discussed "Cost Effective Health Care." Thirty-six representatives of local industry, management, labor, hospitals, and physicians each discussed the problems from their perspective. This was a very fruitful dialogue which is to be expanded in the future to include the insurance industry representatives.

November 1979:

Senator Nancy Kassebaum, who was to speak to MSSC members, was rescheduled for 1980. Coach Gene Smithson presented an entertaining look at 1979-80 Wichita State University basketball.

The MSSC Board approved a request to support an application for certificate of need for the development of the Burn Center at St. Francis Hospital of Wichita.

December 1979:

Donald Woodruff, M.D., gynecological pathologist, was the featured speaker at the general meeting. His topic was "An Evening With Ogden Nash."

University of Kansas School of Medicine-Wichita was responsible for the December meeting of MSSC. There continues to be an excellent relationship between UKSM-W and MSSC. Dean Richard Walsh, M.D., is to be commended for his role in this relationship.

MSSC adopted a policy statement for laboratory service billings.

The Public Relations Commission published in the *MSSC News* a full, double-page patient information sheet explaining reasons for rising health care costs and everyone's role in keeping health care costs down. These are to be available for physicians' office waiting rooms.

Congratulations were extended to Dee Cauble, wife of W. G. Cauble, M.D. She was named "Kansan of the Year" for 1979 for her outstanding work in international health.

Lew W. Purinton, M.D., will become president of MSSC in January 1980.

IVAN E. RHODES, M.D., *Councilor*

DISTRICT 12

Progress has been made in the medical sphere in our district this year. At Pratt, the million dollar expansion of the Pratt Regional Medical Center is rapidly nearing completion. It will vastly expand radiographic facilities, with an active angiography laboratory, relatively sophisticated ultrasound equipment, and a full complement of nuclear scanning capabilities.

Four new physicians have begun practice in our district. Additions of nuclear medicine and ultrasonographic capabilities have increased effectiveness of medical management in many of the other cities as well.

Physicians boarded in the specialties are practicing in the district. Continuing postgraduate training continues to be available to our district members at Harper.

P. J. Antrim, M.D., *Councilor*

DISTRICT 13

Central Kansas Medical Society membership now numbers 68 of whom 79 per cent hold AMA membership. New members include: Richard L. Rajewski, M.D., who joined the Hays Family Practice Clinic; and David F. Harris, M.D., at the Hays Pathology Laboratory. Elected officers for this year include Ward Newcomb, M.D., President; Earl Carlson, M.D., Vice President; Ted Applegate, M.D., Secretary-Treasurer; and L. William Halling, M.D., Alternate Councilor.

We were saddened by the death of one of our active members, Dr. Norman E. Hull.

A CAT scanner is now located in the district at the Hadley Regional Medical Center in Hays. This addition — along with the linear accelerator therapy unit already in operation at St. Anthony's Hospital, also in Hays — allows for more diagnostic as well as therapeutic capabilities at the local and sub-regional levels. This should servc to make western Kansas more attractive to qualified competent physicians, and will also save on commuter energy costs to the patients and families served.

Our area of rural Kansas has been medically underserved. Various programs to help us in this regard continue to be suggested. Latest in this series may be the independent nurse practitioner concept. This apparently would provide a certified, registered, or otherwise approved individual who could also bill directly for certain services rendered. Some of us question if this is truly directed toward a greater availability of qualified care or is simply another stop-gap attempt at demonstrating a lesser cost per "consumer-provider encounter" in the true "lowest bidder" tradition.

Donald D. Goering, M.D., President of the Kansas Medical Society, was guest speaker at our fall quarterly meeting. His observations, repeated at many similar meetings throughout the state, should remind us that there is potential for impact even at the component society level. This impact needs to be directed toward the government and third-party carriers. Failure on our part to join together to express and encourage implementation of our views will only allow the further intrusion of an Orwellian influence upon the private practice of medicine.

Wallace N. Weber, M.D., *Councilor*

DISTRICT 14

Dr. Donald D. Goering, President of the Kansas Medical Society, met with members of District 14 at the Highland Manor Motel in Great Bend on February 20, 1980. He discussed legal problems of the Society and other items of importance to physicians in this area.

Dr. Ralph Reed, Director of the Preceptorship Program at the University of Kansas School of Medicine, and Dr. Fred Whitehead, his assistant, recently discussed plans and projections for the program in District 14. Current preceptees were guests at the meeting and were introduced to members.

Resolutions to be introduced at the annual state meeting in May have been discussed. Members of the district are urged to contact their representatives and senators regarding legislation pending in 1980.

Donald E. Beahm, M.D., *Councilor*

DISTRICT 16

For the first time in a number of years, growth in the Northwest Kansas Medical Society slowed to a standstill. During the year two physicians left the area and we welcomed one new physician, George Kivika, who opened practice in the Rawlins County Clinic in Atwood.

Physicians from Quinter, St. Francis, Colby, Goodland, Hoxie, Oakley, Atwood, Oberlin, and Norton continued to participate in the UKSM Preceptorship Program. Residents from St. Joseph's Family Practice Program, Wichita, continued doing two-month rotations through the Colby Clinic.

President Goering met with us at our October

meeting, where he updated us on the activities of the state society.

Our biggest challenge for 1980 will be to again intensify the recruitment of physicians for the nine northwest Kansas counties that make up our Northwest Kansas Medical Society.

FLOYD L. SMITH, M.D., *Councilor*

DISTRICT 17

There have been few problems and little medical society activity in Council District 17 this past year. The Southwest Kansas Medical Society held only two meetings for reasons that are unclear. Officers for 1980 are: Frank Eichhorn, M.D., President; John Gilbert, M.D., Vice President; and Luther Fry, M.D., Secretary-Treasurer.

Southwest Kansas continues to attract new industry and is one of the more rapidly growing areas of the state. We have been fortunate in adding new physicians to our area to keep pace with this growth. Two family practice physicians have been added in Garden City: Robert Fenton, M.D. and Thomas Koksal, M.D. Dr. Fenton returned following a two-year teaching experience at the University of Nebraska. Two specialists were also added in Garden City: Lauren Welch, M.D., Surgery, and Maura Welch, M.D., Obstetrics-Gynecology. Eva Vachal, M.D. is now our pathologist in Garden City, replacing Dr. Kenneth Heard, who passed away last April. Virgiligo Taduran, M.D. opened his general practice in Satanta; Dr. Dan Dunn has located in Scott City, and Dr. Michael D. Spence in Dighton. Galen Fields, M.D., of Scott City, began his well-deserved retirement in December 1979.

We look forward to 1980 for the challenges and opportunities that only a rapidly developing area can offer.

MAX E. TEARE, M.D., *Councilor*

DISTRICT 18

Council District 18 has added two new physicians: Drs. James Blankenship and Scott Corder are now in family practice in Ottawa.

A new radioisotope scan service has been developed in Garnett and Ottawa sponsored by Research Hospital in Kansas City. This service has been valuable and well-received in this area. Negotiations are underway for the Ottawa ambulance service to be moved to the local hospital.

The Auxiliary spent many months sponsoring and participating in a Follies program to raise money for the monitoring equipment for the hospital operating rooms. A professional director was hired to assist and supervise, and the program was a great success.

In Lawrence, Drs. Gerald Pees and Greg Schnose have established a practice of internal medicine. Dr. Victoria Kimbrough retired this year from the practice of ophthalmology. The local medical society continued to refine the ambulance service, and we are now fortunate to have EMIC technicians and full telemetry service available on the ambulance units. The telemetry is supervised from the emergency room and intensive care units at the hospital.

The Society also helped sponsor the Health Care Conference at the University of Kansas. Dr. Don Hatton ably moderated the event. Interviews were conducted by the Society as part of its participation in the Mediserve program this year. This program is in conjunction with the Farm Bureau.

Much time and discussion were devoted during the year to the development of birthing room facilities at Lawrence Memorial Hospital. This situation has created considerable controversy, but is still under advisement and study.

ROBERT W. HUGHES, M.D., *Councilor*

DISTRICT 19

Council District 19 has maintained its high level of activity throughout 1979. This multiple county district has held monthly meetings at Independence under the leadership of President William Emmott, M.D. The meetings have been well attended, primarily due to the fact that they have been held in conjunction with teaching seminars provided by the University of Kansas School of Medicine. Dr. Emmott has ably directed the Southeast Kansas Medical Society and arranged for the postgraduate seminars at the same time.

One concern of Council District 19 has been the number of members who do not wish to participate in organized medicine. This is on the agenda for further discussion at the next meeting.

State President Donald D. Goering addressed the Southeast Kansas Medical Society on March 19. Topics of some concern to the members in recent months have been the Physician's Assistant legislation and the changes advocated by the American Nursing Association regarding nurse clinicians. The general consensus seems to be that now "everybody wants to play doctor!"

ROBERT F. MOORE, M.D., *Councilor*

Committee and Commission Reports

Actions of KMS Committees and Commissions

CONTINUING MEDICAL EDUCATION

The Continuing Medical Education Committee has been actively participating in resurveys of accredited institutions this past year. Presently there are 25 institutions and organizations in Kansas that are accredited to sponsor Category I credit programs. Twenty-two of these organizations have been surveyed and accredited by the CME Committee. The other three organizations are national in scope and have been surveyed and accredited either by the AMA or the Liaison Committee on Continuing Medical Education (LCCME).

At the national level, the AMA has withdrawn from the LCCME and resumed accreditation activities on its own. Both the AMA and the LCCME plan to survey and accredit organizations of a national scope for Category I credit. Both the AMA and the LCCME have recognized the state medical societies as the accrediting bodies for intrastate programs. There is presently considerable confusion and turmoil over the existence of two national accrediting bodies. The CME Committee is hopeful that the issues resulting in the withdrawal of the AMA from the LCCME will be resolved in the near future. The Kansas State Board of Healing Arts has adopted the position of accepting CME programs from both the AMA and the LCCME; so at present, CME programs for relicensure have not been affected.

For myself and Mr. Caruthers, I would like to thank the KMS for sending us to the AMA Continuing Medical Education Conference in Chicago in October and the LCCME Conference in Phoenix in January. Both conferences were valuable learning experiences on the current status of CME on the national level.

ERWIN JANSSEN, M.D., *Chairman*

DRUG UTILIZATION REVIEW PROJECT

The Kansas Drug Utilization Review Program involves an ongoing review of drug usage patterns for the Kansas Medicaid population. The program is administered by the Kansas Pharmacy Foundation, and functions through a DUR Evaluation Committee chaired by Dr. Wayne O. Wallace, Jr. of Atchison, Kansas. The basic goal of this committee is to assure that patients receive the highest quality of drug therapy at the most economical cost. The accomplishment of this goal is being pursued primarily through educational activities.

During 1979, the committee initiated the *Kansas D.U.R. Newsletter*, which provides information about drug usage in the state to physicians and pharmacists in Kansas. This educational newsletter includes articles on drug therapy and reports on the most frequently prescribed drugs in the Medicaid program and is published monthly. Information for the newsletter is provided through drug usage frequency analysis reports provided by EDS-Federal. Feature articles are developed based on the review of these reports, and from input of members of the Evaluation Committee.

The committee also went through a reorganization of the therapeutic classification system for the Medicaid program. This process was undertaken to provide the DUR Evaluation Committee with a more workable system of drug usage review.

The major activity of the committee focused on the development of a workable method of obtaining diagnosis information as it relates to the drug therapy prescribed. By knowing the intended use of a prescribed drug, a determination of the quality of the drug therapy can be made. Patterns of over-utilization, under-utilization, and inappropriate utilization of drugs are readily identifiable when the diagnosis is known. The committee conducted a pilot project utilizing a diagnosis-coded prescription blank and a speed claim form in order to determine the feasibility of collecting this type of data. Results of that pilot project will be reviewed and evaluated in the coming year.

Other ongoing activities of the committee include the development of patient education messages which are mailed out to Medicaid patients along with their medical cards at different times during the year. Another area of education that is being developed will involve seminars for physicians, pharmacists, and possibly even the public with regard to appropriate drug therapy. The development of topics will be based on data obtained through the review of drug usage in addition to the computer reports obtained from EDS-Federal. Drug utilization in nursing homes is being reviewed and evaluated by the com-

mittee, as this part of the Medicaid population receives a large amount of drug therapy.

WAYNE O. WALLACE, JR., M.D., *Chairman*

EMERGENCY MEDICAL SERVICE

The Emergency Medical Service Committee of the Kansas Medical Society has met on one occasion during this past year. Items discussed at that meeting included prehospital education needs of Kansas, categorization of emergency care facilities, and the role of the Kansas Medical Society in medical control of prehospital care. The committee reviewed the plans for establishment of critical care committees in the areas of burns, trauma, neonatal, poison, cardiac, spinal cord injuries, and behavioral emergencies. The need for establishment of transfer protocols was reviewed and the concept approved by this committee.

The committee remains supportive of emergency medical service efforts in Kansas and wishes to indicate its willingness to provide medical review at the state level.

ERNEST L. MCCLELLAN, M.D., *Chairman*

IMPAIRED PHYSICIANS PROGRAM

Since the first report of this committee given on September 16, 1979, the following activities have occurred:

Records

Committee members have continued to assess calls concerning allegedly impaired physicians. During its January 1980 meeting (which was conducted via telephone conference call), the committee decided that certain bare statistical data needed to be kept if any kind of evaluation of the program is to be possible. The issue of confidentiality was again stressed. With this guiding principle in mind, it was determined that the following information would retain the necessary confidentiality but would provide some idea of the functioning of the committee. Specifically, names would not be recorded. What would be recorded is as follows:

1. The nature of the complaint;
2. The action taken by the committee member;
3. Whether the contact was made directly by the allegedly impaired physician or through a third party;
4. Date of the contact; and
5. Committee member's name.

Suitable forms were prepared and distributed by the Executive Office and mailed to every member of the committee.

Recruitment of Confronters/Managers

An Ad Hoc Committee of three was appointed to consider the question of attracting others to work for the committee — particularly those known to have a particular interest in the area of impairment. This committee has met. Several interested physicians have been contacted. The purpose is to eventually build a resource pool of such persons throughout the state.

Outreach

The Kansas District Branch of the American Psychiatric Association has asked this committee to present a program at the KMS Annual Meeting. A program has been planned and is titled, "The Physician's Work, Marriage and Family: Impairment vs Satisfaction." This will be held on May 1, 1:00-5:00 p.m., at the Shawnee Mission Medical Center.

Education

An Ad Hoc Committee of two was appointed to study the feasibility of more actively introducing the problem of physician impairment and strategies for prevention into the medical curriculum as an elective.

Family Support Program

The committee has enthusiastically endorsed a proposal that a family support program for spouses and families of the impaired physicians be formed. The KMS Auxiliary will assume leadership in this effort and regular announcements will be placed in *Communique*.

Treatment Centers

The committee has identified appropriate centers throughout the State of Kansas. This information is available through the KMS Executive Office in Topeka.

Videotapes

The workshop held on April 28, 1979, which was devoted to techniques of confrontation and motivation, was videotaped. These videotapes are in the process of being edited with the idea that the final edited and short version will be available as an educational tool for appropriate groups of the Kansas Medical Society.

Agenda for the coming months

—Continue development of the statewide network of

contacts/support physicians who can serve as confronters, motivators, and monitors of individual programs;
—pursue the feasibility of developing a workshop for hospital administrators; and
—support and encourage the continued efforts of the ad hoc committees on medical student education, Auxiliary family support groups, and enlistment of contacts/support physicians throughout the state.

GEORGE M. PENN, M.D., *Chairman*

INSURANCE AFFAIRS

The main emphasis of the Insurance Affairs Committee this year was review of its programs. The life insurance, disability benefits, health insurance, and collection programs were all carefully evaluated.

At this time, our members have little interest in group life insurance. It may be that this will be offered again in the future if the need arises. Other programs are functioning well with few problems. The committee urges members of the Kansas Medical Society to contact Gary Caruthers or the chairman of the committee if they have suggestions or complaints. This committee intends to offer to its members only the best insurance programs at the lowest possible price.

RICHARD J. CUMMINGS, M.D., *Chairman*

JOINT KMS-KHA

Your Kansas Medical Society Cost Containment Committee met quarterly during 1979 with representatives from the Kansas Hospital Association, hospital administrators, labor, SRS, business, education and Kansas Blue Cross-Blue Shield. Representing KMS: Phillip A. Godwin, M.D.; Wilbur B. Neel, M.D.; Jerry Slaughter, and myself.

Our efforts focused on the immediate goals of curtailing present costs through correspondence with hospital administrators and medical chiefs of staff encouraging them to look for places to increase efficiency through joint purchasing, elimination of duplication, simplification of drug stocks in pharmacies, attention to the use of the "best buy" in supplies and drugs.

On the long term containment of costs the committee explored methods and ideas to make physicians in training aware of the cost of procedures, equipment, and over-utilization. Pledges from the training programs were sought and in many cases were already in the awareness state of mind. A number of residency programs actually review the hospital costs of selected patients and critique the cost entailed in treatment.

During the present legislative session Mr. Don D. Depew, a Topeka architect, chairman of the Voluntary Effort in Kansas referred to in the first paragraph, appeared before a House Committee to protest the adoption of HB 2750 (the Hospital Cost Containment Act), and to encourage the voluntary effort. He pointed out that the cost of medical care in hospitals and physicians' offices had risen more slowly than the rate of inflation during 1979.

The Voluntary Effort Committee plans to continue to monitor costs and encourage efficiency in the delivery of health care and to point out that bureaucratic regulations, excessive and duplicative paper work, repetitive inspections, and federal intrusion are not part of the solution but a significant part of the problem.

Your Committee will be happy to elaborate on this brief report at the Annual Meeting of the Kansas Medical Society.

ALEX SCOTT, M.D., *Chairman*

JUDICIAL

The Judicial Committee last met on January 26, primarily to review the organizational structure of the committee, to review those complaints being processed, and to set in motion the necessary legal processes (through Wayne Stratton) for the due process portion of the Judicial Committee function.

Pending a reply from Mr. Stratton, we hope we can soon finalize the due process portion and lay the groundwork for the additional legal support that will be needed from the state legislature similar to what was obtained for the Impaired Physician Program.

By the time of State Meeting in May, we hope to have most of this accomplished, although timing will be all-important in some of these matters.

We appreciate the cooperation and support of the KMS membership.

FRANK G. BICHLMEIER, M.D., *Chairman*

KMS-KAPA (PHYSICIAN'S ASSISTANTS) LIAISON

The committee, initially appointed by President Warren Meyer in 1978, was reappointed by President Goering in 1979. Two of our meetings were held jointly with representatives of the Kansas Academy of Physician's Assistants (KAPA).

During the 18 months of committee activity, existing Kansas statutes pertaining to this classification of health professional were reviewed and compared with similar laws passed in other states. The current AMA delegate position regarding utilization of Physician's Assistants was studied and debated. Alleged problems and complaints voiced in the recent past were investigated and for the most part could not be substantiated.

At its concluding meeting on September 30, 1979, the KMS committee unanimously approved the following statement on supervision to be presented to the Executive Committee:

It shall be the Kansas Medical Society policy that the personal presence by a supervising physician in the Physician's Assistants' activities where the services are rendered should be the standard of care in the usual practice setting. In unusual practice settings, exceptions to the standard of care should be determined and a specific waiver to this standard may be granted by the Kansas State Board of Healing Arts on an individual case-by-case basis, after annual approval by the local county medical society.

The committee further recommended formation of a joint liaison committee (KMS and KAPA) to review alleged problems relating to the utilization of Physician's Assistants by members of the Kansas Medical Society.

D. Cramer Reed, M.D., *Chairman*

KMS-KSNA (NURSES) LIAISON

Because the issue was still being considered at the time the *Journal* went to press, an up-to-date report will be given at the House of Delegates.

KANSAS MEDICAL POLITICAL ACTION COMMITTEE

During the past year, KaMPAC has been actively soliciting contributions. An informational brochure and dues return envelope was developed to provide information and simplify the return of dues. The goal is to establish a substantial fund for the 1980 elections. This year all state senators and representatives are up for re-election as well as one U. S. Senator and the five U. S. Congressmen. The Board of Directors will be meeting later this summer to review all candidates for state and federal office to determine which ones should receive support. If you have a candidate in your local area whom you believe deserves support, please contact a member of the KaMPAC Board of Directors. On behalf of the KaMPAC Board of Directors, I would like to thank the Kansas Medical Society for the contribution of $2,500 to the Educational Fund to assist us in our administrative expenses.

Ronald Davis, M.D., *Chairman*

MENTAL HEALTH

The Department of Social and Rehabilitation Services is steadily reducing the bed capacities of the state hospitals, producing an increasing population of chronically mentally ill persons in the community. For example, the number of adult beds at Topeka State Hospital has been reduced from 750 to 160. Most of these 500 displaced persons end up in nursing homes (which are designed to serve primarily geriatric and other physically handicapped people) and problems are rife. The Department of Health and Environment is receiving considerable protest from the nursing homes. Our committee is working with the Kansas Psychiatric Society's Committee on Mental Health Services to see what can be done to improve the situation. In the near future, the committee may have a recommendation to propose for consideration by the House of Delegates.

A bill in the legislature proposes to license master-degreed (M.A.) psychologists for independent, private practice. In one sense, this issue is an intra-professional one for the state's psychologists since the present law permits Ph.D. psychologists independent practice for psychotherapy and counseling without the need for medical supervision or cooperation. However, reducing the requirements to permit considerably less qualified psychologists to treat distressed clients is open to question. The Kansas Psychiatric Society is taking a stand against the bill, and this seems an appropriate action for them. This committee does not recommend Medical Society action at this time.

The Mental Health Committee endorsed Senate Bill 444 providing that pre-school child health assessments — now available only to children of Medicaid families — be extended to all children in Kansas. The endorsement indicated that this action was from the committee only, and did not constitute official sanction of the Medical Society.

Herbert Modlin, M.D., *Chairman*

NECROLOGY REPORT

Following is a list of the members of the Kansas Medical Society whose deaths have been reported since the last meeting of the House of Delegates.

Name and Address	*Age*	*Date*
Rene A. Alonso, *Topeka*	58	Oct. 11, 1979

John H. Basham, *Eureka*	67	April 23, 1979
Harold Bowman, *Wichita*	75	Oct. 6, 1979
Richard H. Clairborne III, *Baxter Springs*	51	June 16, 1979
George E. Cowles, *Wichita*	83	Dec. 7, 1979
Murray C. Eddy, *Hays*	81	April 30, 1979
Alfred S. Hawkey, *Newton*	75	May 16, 1979
Charles E. Henneberger, *Atwood*	93	Aug. 28, 1979
John D. Huff, *Kansas City*	57	Sept. 16, 1979
Norman E. Hull, *Hays*	55	Jan. 3, 1980
Joseph H. Johnson, *El Dorado*	80	Sept. 27, 1979
Henry W. Kassel, *Kansas City*	73	Feb. 28, 1979
Irene Koeneke, *Halstead*	81	July 12, 1979
Eugene J. McCreight, *Liberal*	79	Oct. 10, 1979
W. L. Pratt, *Leavenworth*	74	April 27, 1979
Robert G. Rate, *Halstead*	65	Oct. 25, 1979
Albert J. Rettenmaier, *Kansas City*	84	Nov. 26, 1979
Edward J. Ryan, *Emporia*	67	Dec. 14, 1979
Glen R. Shepherd, *Kansas City*	59	Sept. 1, 1979
Galen M. Tice, *Kansas City*	80	Sept. 22, 1979
Eugene A. Walsh, *Onaga*	61	Nov. 5, 1979
Gene V. Williams, *El Dorado*	55	July 4, 1979

NOMINATING

The Nominating Committee of the Kansas Medical Society submits the slate of candidates for the offices of the Society, to be voted on by the House of Delegates at the Society's Annual Meeting May 2-4, 1980, at Kansas City, Kansas.

President Elect

Herman W. Hiesterman, M.D., Quinter. Born in 1923. Graduated from the University of Kansas School of Medicine in 1951. Is in Family Practice. Is currently serving as First Vice President.

First Vice President

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Is now serving as Second Vice President and AMA Alternate Delegate.

Second Vice President

Richard L. Brownrigg, M.D., Dodge City. Born in 1935. Graduated from the University of Kansas School of Medicine in 1961. Practices Urology. Is currently serving as Councilor for District 15.

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now serving as Chairman of the Legislative Committee.

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is now Chairman, Commission on Education, and AMA Alternate Delegate.

Roger D. Warren, M.D., Hanover. Born in 1931. Graduated from the University of Kansas School of Medicine in 1957. Practices General Surgery. Is currently serving as Councilor for District 1.

Constitutional Secretary

Jack R. Cooper, M.D., Shawnee Mission. Born in 1917. Graduated from Ohio State University School of Medicine in 1943. Practices Neurosurgery. Is currently serving as Secretary.

Treasurer

William K. Walker, M.D., Sedan. Born in 1928. Graduated from the University of Kansas School of Medicine in 1945. Is in Family Practice. Is now serving as Treasurer.

Speaker

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and Speaker of the KMS House of Delegates.

Vice Speaker

John O. Yulich, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1959. Is in Family Practice. Now serves as Vice Speaker of the KMS House of Delegates.

AMA Delegate

Alex Scott, M.D., Junction City. Born in 1923. Graduated from the University of Wisconsin Medical School in 1948. Is in Family Practice. Is serving as AMA Delegate.

AMA Alternate Delegate

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Is currently serving as Second Vice President and AMA Alternate Delegate.

The submission of the slate completes the work of the Committee and the slate serves as the report of the Committee to the House of Delegates.

Thank you for the privilege of serving the Society.

WARREN E. MEYER, M.D., *Chairman*

PROFESSIONAL LIABILITY COMMISSION

The Commission on Professional Liability introduced four bills in the last session of the Legislature that were designed to make significant improvements in the professional liability situation in Kansas. The bills dealt with expert witnesses, establishing a standard of care, requiring a notice of intent to file suit, limiting oral guarantees of favorable results, and clearly establishing confidentiality of certain hospital committee proceedings. Of the bills, the one that would have guaranteed confidentiality of staff committee proceedings was by far the most important and needed. Unfortunately, all the bills were referred to the House Judiciary Committee, where it is virtually impossible to pass anything that resembles constructive tort reform. The Commission is now working to revitalize a bill for consideration next year on the confidentiality of staff committee proceedings. We are meeting with hospital representatives, attorneys, and others to develop a bill that reflects our major concerns and at the same time has a reasonable chance of passing the legislature.

The Commission has a full agenda for the coming year, with primary emphasis on investigating risk

management programs. At its April meeting, the Commission will hear from a nationally known authority and begin its review of programs that may be feasible for implementation in Kansas. Representatives of the Hospital Association and the Insurance Commissioner's office will be asked to participate in our proceedings as the year goes on, and the Commission hopes to have a definite proposal before the year is over.

The Commission hopes that individuals with suggestions, comments, problems, or questions will contact the members at any time during the year, so that any areas of concern can be properly addressed. Your input is vital and we welcome it.

DANIEL K. ROBERTS, M.D., *Chairman*

SCHOOL HEALTH

The School Health Committee has not met formally this past year. Committee members have, however, maintained contact.

One of the problems discussed was the problem of school health physicals done by school health nurses. The general consensus was that they should not conduct them unless they had special training for this. In view of other action taken on this, our committee felt that we need not take further action at this time.

WILLIAM NICE, M.D., *Chairman*

REHABILITATION

The Committee on Rehabilitation met in October 1979 to discuss the report of the Emergency Medical Services Spinal Cord Injury Critical Care Committee. This committee, headed by Gregg M. Snyder, M.D. — in response to Senate Bill 248 (1978) — had prepared a report directed to the Secretary of Health and Environment relating to the establishment of a multi-level treatment program for persons with spinal cord injuries. Dr. Snyder's report was favorably received by the members of this committee. Basically there are three recommendations:

1. The formation of a State of Kansas Spinal Cord Injury Registry;
2. A planning system of levels of care for such patients including naming several centers for comprehensive care to serve the eastern and western parts of the state: and
3. The development of a post-discharge program of follow-up for patients who have been through the comprehensive programs so that their lifetime needs in health care can be properly determined.

This report is currently in the hands of the State Health Department where a plan is evolving to collect, process, and evaluate data pertaining to all phases of spinal cord injury and treatment for the ultimate purpose of designating a system of spinal cord injury rehabilitation treatment in Kansas.

The other major action of the committee was to provide the Kansas Foundation for Medical Care, Inc., with guidelines for admission and treatment of patients in rehabilitation centers as required by the PSRO review process. This report was circulated to the membership of the committee and approved by the majority of the committee as the official stand in regard to PSRO guidelines. Basically, the report recommends that all rehabilitation units should have a regularly scheduled team conference headed by a physician where goals for each patient must be carefully documented and outlined. Evaluation as to whether the goals of rehabilitation were met should be part of the record of every rehabilitation patient. All patients in rehabilitation centers should have a functional assessment and require more than one service to be performed to meet that functional goal. In other words, if all that is offered is physical therapy, for example, this cannot be considered an adequate description of a medical rehabilitation program. A rehabilitation program must be medically directed and properly coordinated.

J. B. REDFORD, M.D., *Chairman*

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Resolutions

To Be Introduced at First House of Delegates, May 3, 1980

REFERENCE COMMITTEE

J. Rex Stone, M.D., Manhattan, *Chairman*
Richard Meidinger, M.D., Topeka
William W. Burney, Sr., M.D., Wichita
J. Roderick Bradley, M.D., Greensburg
John W. Young, M.D., Shawnee Mission

An asterisk following the resolution number indicates that those resolutions require a change in the Constitution and By-Laws, and a two-thirds majority vote of the House of Delegates is needed.

RESOLUTION NO. 80-1

(Submitted by Physician's Assistant Liaison Committee)

Physician's Assistants — Supervision

Resolved, That it shall be the Kansas Medical Society policy that the personal presence by a supervising physician in the Physician's Assistant's activities where the services are rendered should be the standard of care in the usual practice setting; and be it further

Resolved, That in unusual practice settings, exceptions to the standard of care should be determined and a specific waiver to this standard may be granted by the State Board of Healing Arts on an individual case-by-case basis, after annual approval by the local county medical society.

RESOLUTION NO. 80-2

(Submitted by the Executive Committee)

Chiropractic

Resolved, That the diagnosis and treatment of disease by practitioners attributing all such ailments to misalignment of the spinal vertebrae is a hazard to health; and be it further

Resolved, That a patient who relies on such practitioners in the treatment of ailments such as essential hypertension, heart disease, stroke, cancer, diabetes, and infections may delay proper medical care until serious and irreversible damage occurs.

RESOLUTION NO. 80-3*

(Submitted by the Executive Committee)

Official Policy of the Kansas Medical Society

Resolved, That official policy on a given subject established through resolutions at the House of Delegates shall be the most recent action taken on that subject. All other prior resolutions dealing with that subject shall be rescinded and deleted as per Resolution No. 66-43; and be it further

Resolved, That the bylaws be amended with the addition of Section 5.444 *Expiration:* Official policies established through resolutions at the House of Delegates shall be in effect for a period of five (5) years at which time that policy position will expire unless superseded or continued by another resolution.

RESOLUTION NO. 80-4

(Submitted by the Continuing Medical Education Committee)

Continuing Medical Education Accreditation

WHEREAS, The Kansas Medical Society CME Committee is interested in an active forum for the sharing of CME accreditation information among all the state medical societies; and

WHEREAS, The Kansas Medical Society CME Committee is concerned with the present turmoil surrounding the withdrawal of the AMA from the Liaison Committee on Continuing Medical Education and the subsequent accreditation activities by both organizations, therefore be it

Resolved, That the AMA instruct the Department of Continuing Medical Evaluation to provide regular opportunities for state CME chairmen and staff to meet and interact with AMA continuing medical education staff at the annual AMA/CME conference; and be it further

Resolved, That the AMA be encouraged to explore all possible avenues for the establishment of one national accrediting body.

RESOLUTION NO. 80-5

(Submitted by the Executive Committee)

Kansas Voluntary Effort

WHEREAS, The Voluntary Effort on Cost Containment has shown its ability to restrain the amount of increase in health care costs; therefore be it

Resolved, That the Kansas Medical Society reaffirm its support for the Voluntary Effort on Cost Containment.

RESOLUTION NO. 80-6

(Submitted by the Butler-Greenwood Medical Society)

Voluntary Effort

WHEREAS, The legislature will look at prospective hospital rate and budget review during the coming legislative session; and

WHEREAS, There are a variety of different state mechanisms already in existence; some voluntary (Indiana), some legislated (Maryland, Washington State) with some questions about amounts actually saved, and some whose power is being challenged in the courts; and

WHEREAS, The voluntary effort on cost containment has shown its ability to restrain the amount of increase in health care costs and has the approval of physicians, hospitals and government; and

WHEREAS, The voluntary cost containment effort encompasses a much wider scope than the hospital rates in its combined effort; therefore be it

Resolved, That the Kansas Medical Society endorse the Kansas Voluntary Effort for Cost Containment and petition the Kansas Legislature to defer action on any legislated hospital rate review program pending further information on the continuing effectiveness of this effort.

RESOLUTION NO. 80-7

(Submitted by the Executive Committee)

Kansas Hospital Prospective Rate Review

Resolved, That the Kansas Medical Society endorses the Kansas Hospital Prospective Rate Review Program as sponsored by Blue Cross.

RESOLUTION NO. 80-8

(Submitted by the Executive Committee)

Legislative Hospital Rate Review

Resolved, That the Kansas Medical Society opposes any type of legislated hospital rate review.

RESOLUTION NO. 80-9

(Submitted by the Executive Committee)

Principles of Medical Ethics

Resolved, That the revised version of the Principles of Medical Ethics as proposed by the Ad Hoc Committee of the AMA be endorsed by the Kansas Medical Society; and be it further

Resolved, That the Kansas delegates to the AMA House of Delegates be instructed to vote in favor of adoption of the revised version of the Principles of Medical Ethics.

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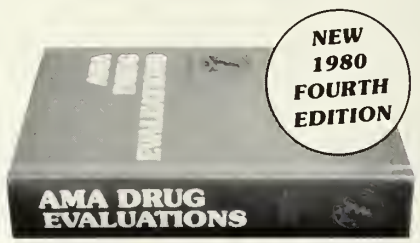
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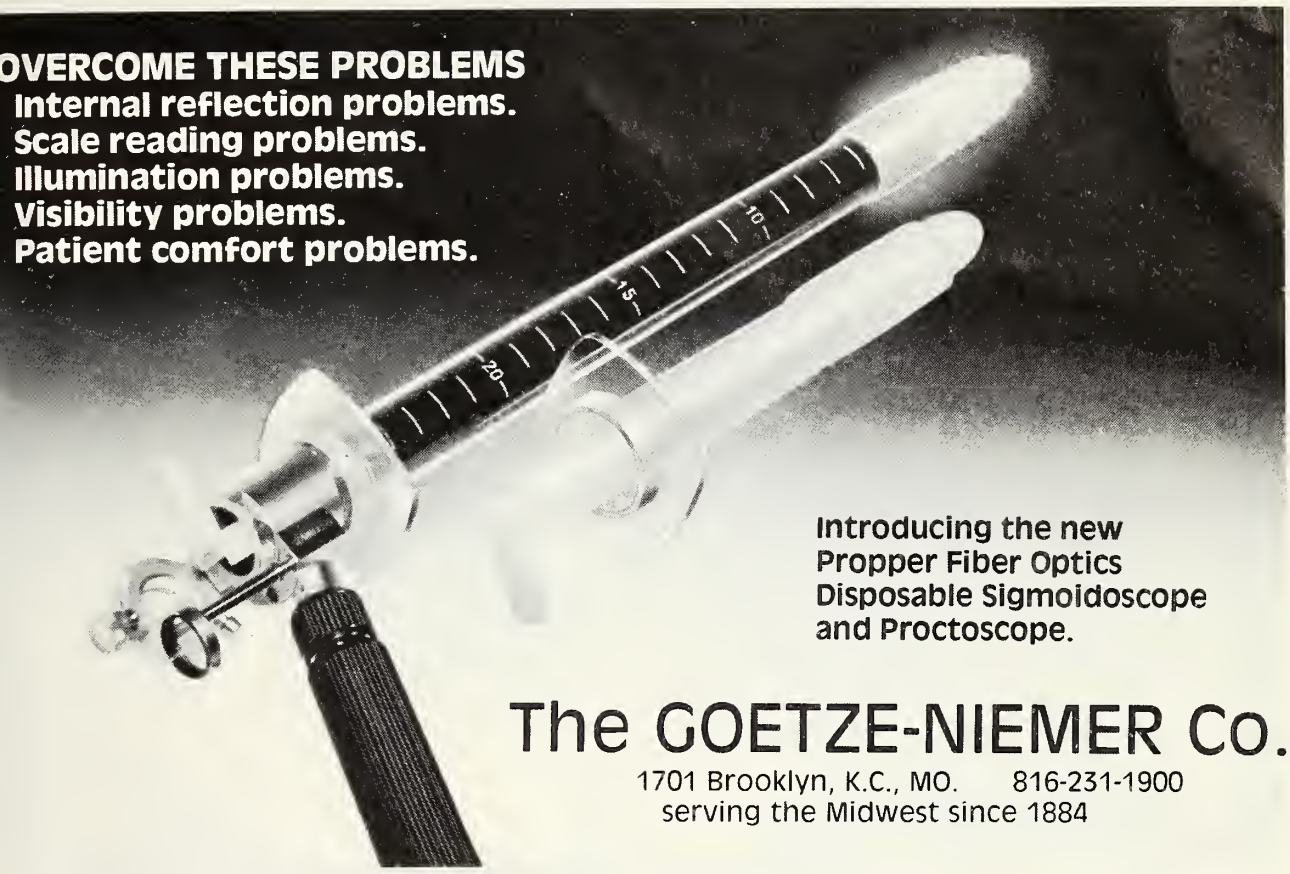
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FRIDAY, MAY 2

9:00 Pre-Convention Board of Directors Meeting
Mrs. Kermit G. Wedel, Minneapolis, President
The Kansas Medical Society Auxiliary, Presiding

12:00 Luncheon Honoring Past Presidents

1:30 General Session
Mrs. Kermit G. Wedel, Presiding

7:00 AMAERF Auction, KMS and KMSA

SATURDAY, MAY 3

7:00 Past Presidents' Breakfast

9:00 KMS Opening House of Delegates
(KMSA President reports)

9:30 General Session and Installation of Officers
Mrs. Kermit G. Wedel, Presiding

12:30 Punchbowl
Mrs. Kermit G. Wedel, Presiding

1:00 Luncheon Honoring National President, Mrs. Ben Johnson, Jr.

3:00 Post-Convention Board of Directors Meeting
All new board members, county presidents and presidents-elect are urged to attend.

5:30 KU Alumni Reception

7:00 Annual KMS President's Banquet
The dinner will be followed by a concert and dancing.

PRE-REGISTRATION REQUESTED

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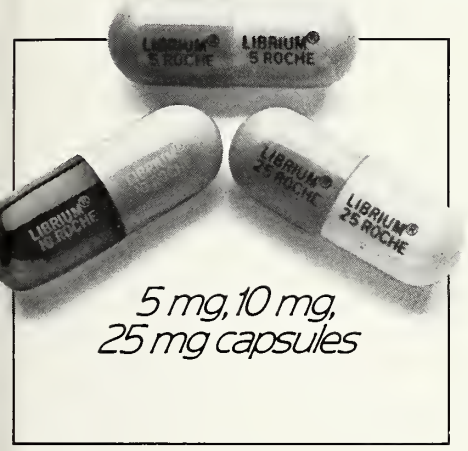
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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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The new roster will be published in August

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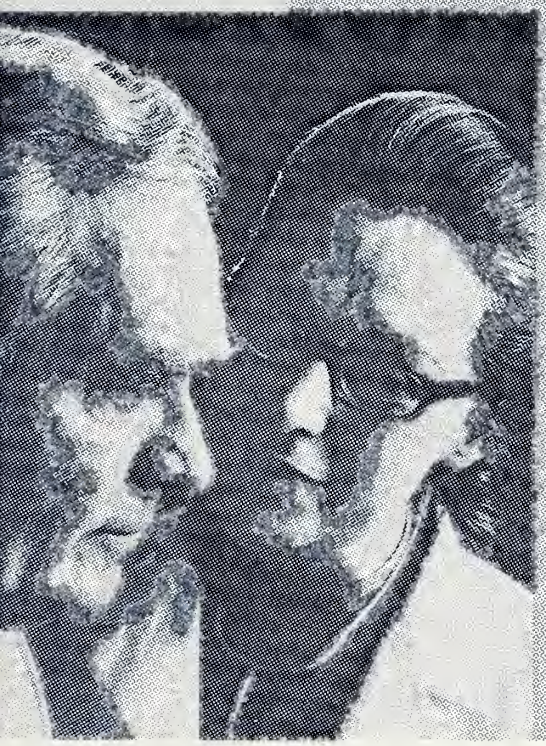
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Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally; making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients*, 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

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Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.



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See next page for brief summary of prescribing information.

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DESCRIPTION

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This product is prepared from human venous plasma. Each individual unit of plasma has been found nonreactive for hepatitis B surface antigen using the radioimmunoassay method of counter-electrophoresis.

INDICATIONS

Treatment of rabies, once clinical disease becomes apparent, is rarely if ever successful. Rabies vaccine (duck-embryo origin, Lilly Laboratories) with or without Rabies Immune Globulin (Human) – Hyperab® should, therefore be given to all persons suspected of exposure to rabies, particularly to severe exposure. Whenever possible, Hyperab® globulin should be injected as promptly as possible after exposure. If initiation of treatment is delayed for any reason, however, Rabies Immune Globulin (Human) should be given just the same, regardless of the interval between exposure and treatment.

Rabies virus is usually transmitted by the bite of a rabid animal, but can occasionally penetrate abraded skin with the saliva of infected animals. Progress of the virus after exposure is believed to follow a neural pathway, and the time between exposure and clinical rabies is a function of the proximity of the bite (or abrasion) to the central nervous system and the dose of virus injected. The incubation is usually 2 to 6 weeks, but can be longer. After severe bites about the head and neck it may be as short as 10 days.

After initiation of the vaccine series, it takes 2 weeks or longer for development of immunity to rabies. Since most vaccine failures have occurred in cases of severe exposure, the value of immediate immunization with preformed rabies antibody cannot be over-emphasized.

Recommendations for use of passive and/or active immunization after exposure to an animal suspected of having rabies were detailed by WHO, and by the US Public Health Service Advisory Committee on Immunization Practices (ACIP).

INJECTION PROCEDURE

A portion of the Hyperab® globulin dose should be used to infiltrate the wound. The rest is injected intramuscularly.

CONTRAINDICATIONS

Rabies Immune Globulin (Human) – Hyperab® is contraindicated in repeated doses, once vaccine treatment has been initiated. Repeating the dose may bring about interference with full expression of active immunity expected from the vaccine. Hyperab® globulin is also contraindicated in individuals who are known to have an allergic response to gamma globulin or thimerosal.

PRECAUTIONS

NEVER ADMINISTER Hyperab® globulin INTRAVENOUSLY.

ADVERSE REACTIONS

Slight soreness at the site of injection, and slight temperature elevation, may be noted at times. Sensitization to repeated injections of human globulin is extremely rare.

In the course of routine injections of a large number of persons with human gamma globulin, there have been a few isolated occurrences of angioneurotic edema, nephrotic syndrome, and anaphylactic shock after injection. Because of their rarity, it is difficult to determine whether such reactions are incidental, or causally related to the gamma globulin.

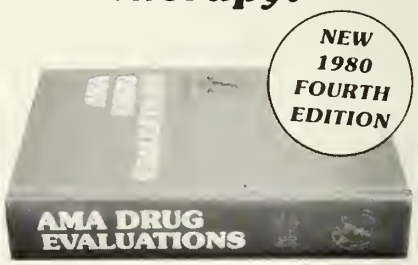
No instances of transmission of hepatitis B (homologous serum jaundice) have been reported from the use of human gamma globulin prepared by the fractionation methods employed by Cutter Laboratories, Inc.

HOW SUPPLIED

Rabies Immune Globulin (Human) – Hyperab® is packaged in 2-ml. and 10-ml. vials with a potency of 150 International Units per ml. (IU/ml.). The 2-ml. vial contains a total of 300 IU which is sufficient for a child weighing 15 kg. (33 lb.). The 10-ml. vial contains a total of 1500 IU which is sufficient for an adult weighing 75 kg. (165 lb.).

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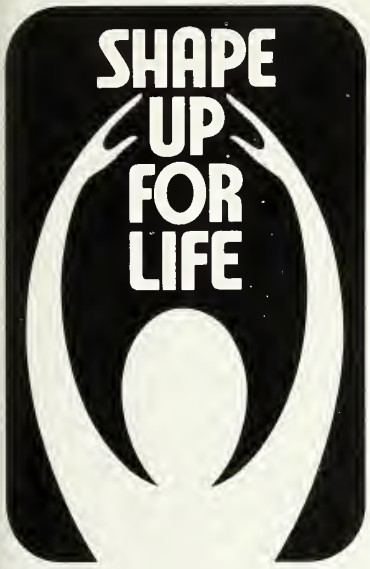
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American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

Auxiliary News

The annual convention of the American Medical Association Auxiliary will convene in Chicago, July 20-23. The increase in dues — from $7 to $11 — will become effective July 1.

A recent pamphlet from the AMAA gives some insight into the structure and programs of the organization.

The American Medical Association Auxiliary is a not-for-profit, volunteer, service organization comprised of more than 81,000 physicians' spouses in 50 states and the District of Columbia. The primary objective is to improve the health and welfare of the people in local communities by working to fill local needs. Auxiliary participation provides unique opportunities for physicians' spouses to become involved in health-related activities, and all current health-related issues are considered in formulating plans for projects.

The Auxiliary works closely with the AMA to help support sound legislation — both federal and state — which affects the health and welfare of people in their communities. Fund raising for grants to medical schools and medical student scholarships and loans is carried on across the country.

Each auxiliary is encouraged to closely examine its local community with regard to existing health services and health needs, then to implement programs to fill the gaps, improve basic programs, or institute a needed community service.

The AMAA stands ready to assist the local organizations with informational materials. A national magazine *Facets*, published quarterly, highlights current health issues and auxiliary programs for every member. In addition, *Direct Line* (bimonthly) gives program information to state and county leaders; and *National News* (bimonthly) explains policy to state leaders. The newest publication, *Horizons*, brings the latest information on auxiliary activities and services to spouses of resident physicians and medical students.

Only physicians' spouses are eligible for membership in the AMA Auxiliary. For further information contact your county chapter, or the KMS office.

PHYSICIAN — M.D. or D.O.

Kansas Social and Rehabilitation Services, Medical Services Section. Position is vacant for a medical doctor or doctor of osteopathy to administer the Hospital and Medically Related Services Unit. Responsibilities include evaluating services being provided by physicians, hospitals, dentists, and other professionals who are providers in the Medicaid (Medical Assistance) Program recommending policy changes, reviewing services for authorization and assisting the Chief Administrator and staff in other ways in administering the programs. Must be interested in public service and developing a strong recipient oriented program. Salary negotiable. Application and résumé should be submitted to:

L. Kathryn Klassen, R.N., M.S.
Chief Administrator
Medical Services Section
Social and Rehabilitation Services
6th Floor, State Office Building
Topeka, Kansas 66612
Tele: (913) 296-3981

Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

Editor
The Journal of the Kansas Medical Society
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CONTRAINDICATIONS Use in Newborn or Premature Infants. This drug should not be used in newborn or premature infants

Use in Nursing Mothers. Because of the higher risk of antihistamines for infants generally and for newborns and prematures in particular, antihistamine therapy is contraindicated in nursing mothers

Use in Lower Respiratory Disease. Antihistamines should NOT be used to treat lower respiratory tract symptoms including asthma

Antihistamines are also contraindicated in the following conditions: hypersensitivity to azatadine maleate and other antihistamines of similar chemical structure; monoamine oxidase inhibitor therapy (See DRUG INTERACTIONS Section).

WARNINGS Antihistamines should be used with considerable caution in patients with: narrow angle glaucoma, stenosing peptic ulcer, pyloroduodenal obstruction, symptomatic prostatic hypertrophy, bladder neck obstruction

Use in Children: In infants and children especially, antihistamines in overdosage may cause hallucinations, convulsions, or death

As in adults, antihistamines may diminish mental alertness in children. In the young child, particularly, they may produce excitation

OPTIMINE TABLETS ARE NOT INTENDED FOR USE IN CHILDREN UNDER 12 YEARS OF AGE

Use in Pregnancy: Experience with this drug in pregnant women is inadequate to determine whether there exists a potential for harm to the developing fetus

Use with CNS Depressants. Azatadine maleate has additive effects with alcohol and other CNS depressants (hypnotics, sedatives, tranquilizers, etc.).

Use in Activities Requiring Mental Alertness. Patients should be warned about engaging in activities requiring mental alertness, such as driving a car or operating appliances, machinery, etc.

Use in the Elderly (approximately 60 years or older): Antihistamines are more likely to cause dizziness, sedation, and hypotension in elderly patients

PRECAUTIONS Azatadine maleate has an atropine-like action and, therefore, should be used with caution in patients with: a history of bronchial asthma, increased intraocular pressure, hyperthyroidism, cardiovascular disease, hypertension

DRUG INTERACTIONS MAO inhibitors prolong and intensify the anticholinergic (drying) effects of antihistamines

ADVERSE REACTIONS The most frequent adverse reactions are underlined

General: Urticaria, drug rash, anaphylactic shock, photosensitivity, excessive perspiration, chills, dryness of mouth, nose, and throat

Cardiovascular System: Hypotension, headache, palpitations, tachycardia, extrasystoles

Hematologic System: Hemolytic anemia, thrombocytopenia, agranulocytosis

Nervous System: Sedation, sleepiness, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesias, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis, hysteria, neuritis, convulsions

Gastrointestinal System: Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation.

Genitourinary System: Urinary frequency, difficult urination, urinary retention, early menses

Respiratory System: Thickening of bronchial secretions, tightness of chest and wheezing, nasal stuffiness

OVERDOSAGE Antihistamine overdosage reactions may vary from central nervous system depression to stimulation. Stimulation is particularly likely in children. Atropine-like signs and symptoms (dry mouth, fixed, dilated pupils, flushing; and gastrointestinal symptoms) may also occur

If vomiting has not occurred spontaneously, the patient should be induced to vomit. This is best done by having him drink a glass of water or milk after which he should be made to gag. Precautions against aspiration must be taken, especially in infants and children.

If vomiting is unsuccessful, gastric lavage is indicated within three hours after ingestion and even later if large amounts of milk or cream were given beforehand. Isotonic and ½ isotonic saline is the lavage solution of choice

Saline cathartics, such as milk of magnesia, draw water into the bowel by osmosis and therefore are valuable for their action in rapid dilution of bowel content.

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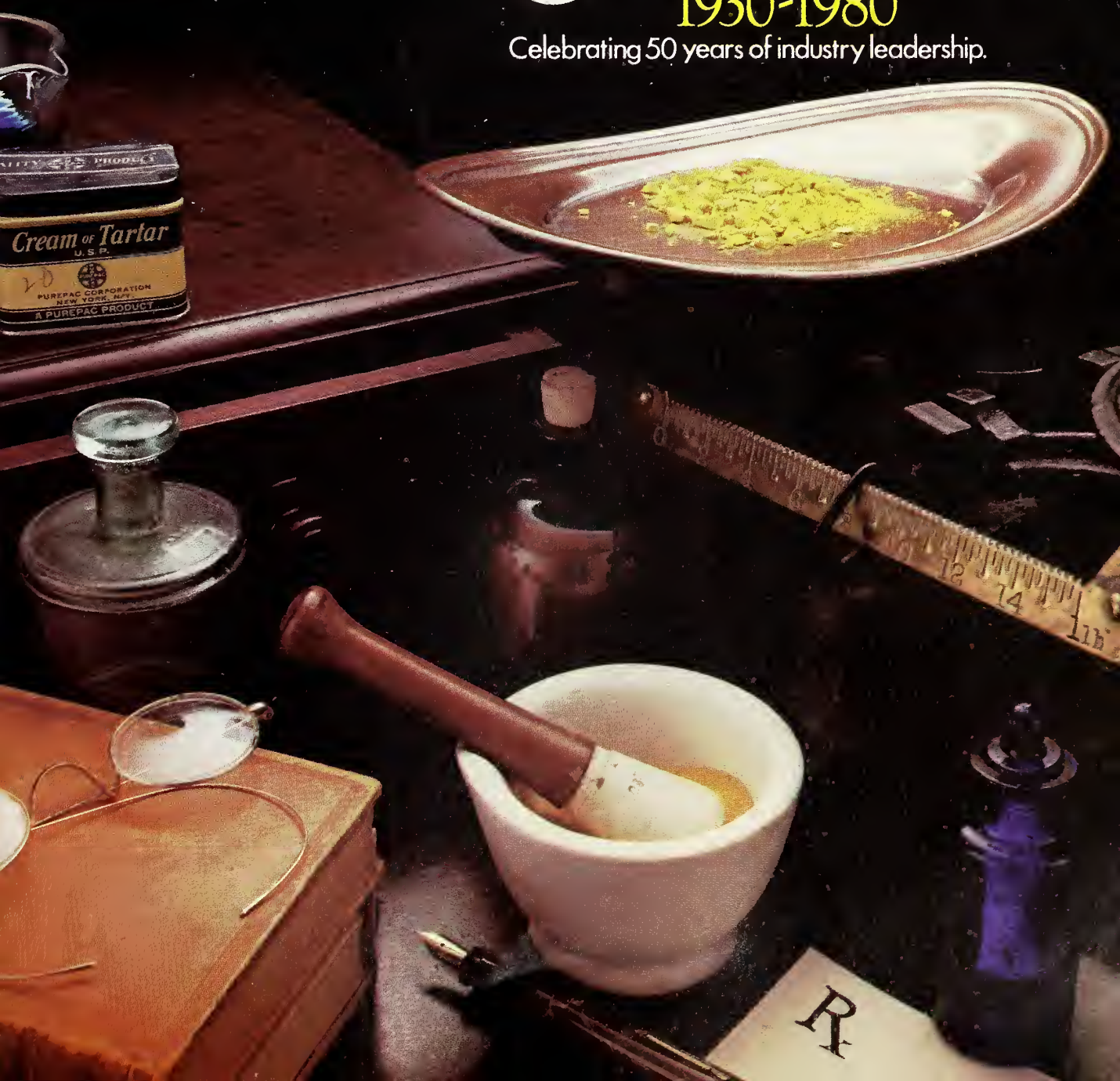
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antianxiety/antisecretory/antispasmodic

for adjunctive therapy of duodenal ulcer* and irritable bowel syndrome*

Librax®

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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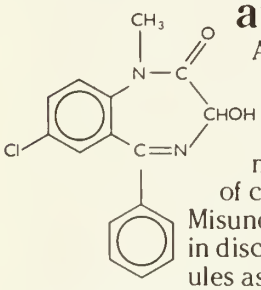
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What to tell your patients when you prescribe Valium® (diazepam/Roche)

Survey shows significant correlation between comprehension and compliance



A study of compliance patterns reveals that more than 6 out of 10 patients made errors in self-administration of prescribed medication, largely due to lack of comprehension.* Misunderstanding of directions resulted in discrepancies in dosage schedules as well as in length of therapy. Since evidence suggests that expanded verbal instructions may encourage compliance, the patient receiving Valium can benefit from your explanation of the dosage regimen, what response to expect from therapy and when to expect it.

What Valium (diazepam/Roche) can do

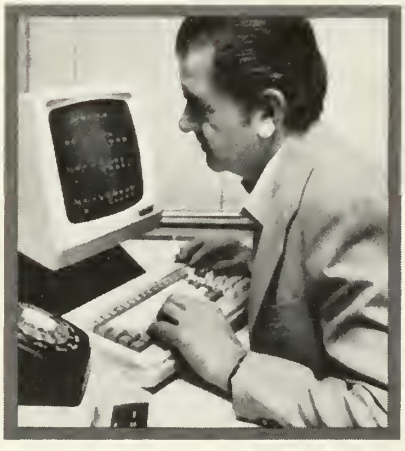
Your patients should know that 1) you are prescribing Valium as an adjunct to an overall program for the treatment of anxiety, and 2) Valium is given to relieve the symptoms of excessive anxiety and psychic tension while you help the patient to explore and deal with the underlying cause of his psychic tension.

Patients often interpret manifestations of anxiety, such as palpitations, hyperventilation, fatigue and muscle tension, as symptoms of a serious disease. However, when they learn that these symptoms can be relieved by Valium therapy, patients can more readily understand the psychosomatic origin of their symptoms and to accept the nonpharmacologic measures you may recommend.

The time you devote to these explanations can be a therapeutic measure in itself. Most anxious patients respond to and benefit from a frank discussion with an objective, sympathetic professional.

At the start of treatment, establishing therapeutic goals helps the patient to learn *what* to expect and *when* to expect it. Patients should also be informed that the medication will be gradually reduced and discontinued upon attainment of the therapeutic goal.

Tapering of dosage is rarely necessary in short-term therapy, but when consistently higher doses are used for extended periods, patients should know that the gradual reduction of medication will be implemented in order to avoid sudden recurrence of symptoms or possible withdrawal symptoms. Such recurrence is unlikely when the causes of the anxiety have been worked out satisfactorily within your overall treatment program.



What Valium (diazepam/Roche) can't do

It should be emphasized that there is no "magic" in any antianxiety tablet; that medication is not prescribed as a problem solver. Instead, Valium is being prescribed *as a temporary measure to relieve symptoms* generated by excessive anxiety and psychic tension.

* Boyd JR, *et al*: *Am J Hosp Pharm 31*: 485-491, May 1974

Before prescribing, please consult complete product information, a summary of which follows:

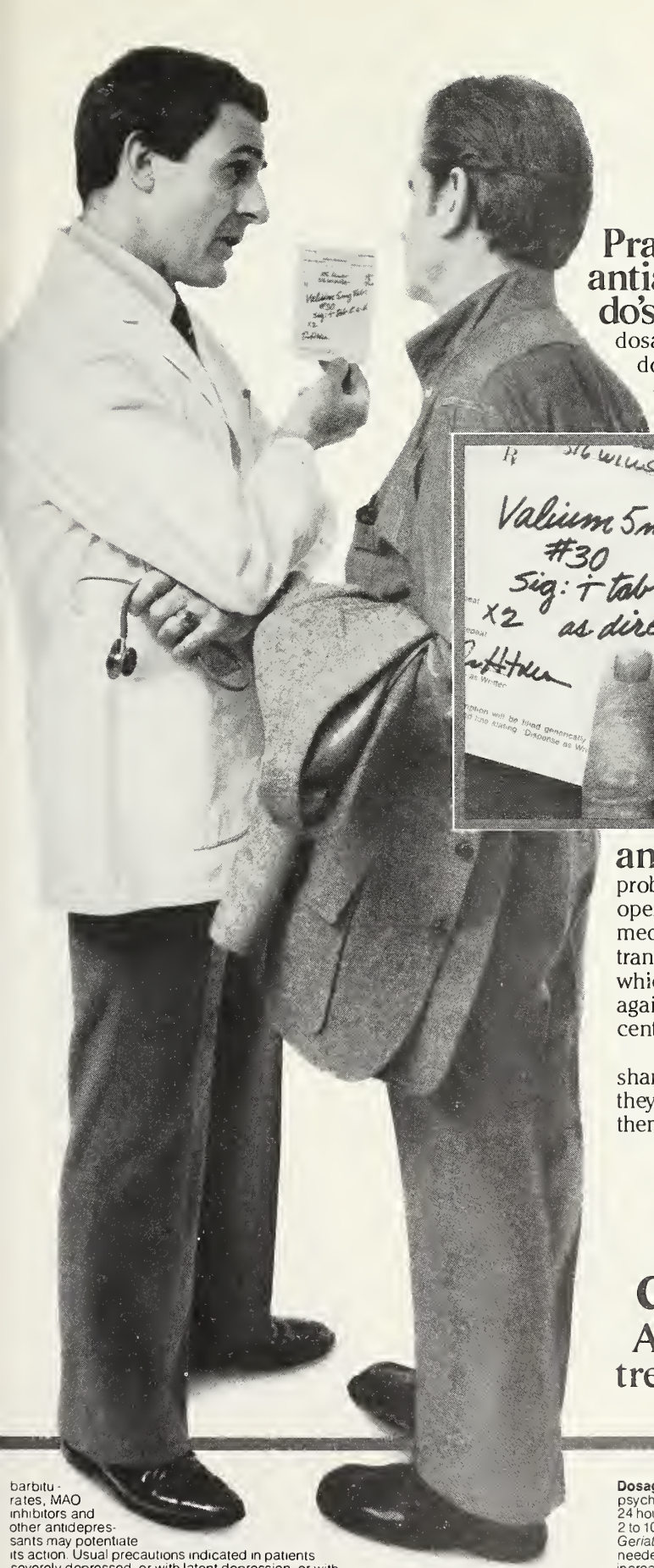
Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders, psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms, or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics,



Practical pointers on taking antianxiety medications

do's Patients should be instructed to keep to their dosage schedule exactly as prescribed. If they miss a dose, they should not try to make it up by taking two doses the next time. Ask them to contact you promptly if they experience worrisome side effects.

Explain that drowsiness is a common reaction to almost all calming agents, but that it usually subsides in a few days. Urge the patient to contact you for a possible dosage adjustment if drowsiness or other reactions persist.

Just as you request a complete list of all medications the patient is taking, suggest that this list be given to any other physician treating her/him.

Like all medicines, Valium should be kept out of reach of children and young people. Old or unused medication should be discarded.

and don'ts Since drowsiness is an occasional problem, patients should be advised against driving or operating hazardous machinery until they see how the medication affects them. They should also know that tranquilizers increase the effects of alcoholic beverages, which should therefore be avoided. Also, warn patients against simultaneous use of drugs that depress the central nervous system, particularly sedative hypnotics.

Patients should be aware of the importance of not sharing their medications with friends and neighbors; they should know that what you have prescribed for them may be contraindicated for others.

2-mg, 5-mg, 10-mg scored tablets

Valium®(IV)
diazepam/Roche

An important adjunct to your treatment program for excessive psychic tension

barbitu - rates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam/Roche) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

ROCHE®

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Symptom: rising administrative costs

Prescription: cost-containing ideas from Southwestern Bell

He's Vice-President for growing Methodist Hospital in Dallas. She's a health care Accour Executive for Southwestern Bell.

And whether she or one of her health care team members is reviewing procedures in the admissions area or consulting with hospital administrators, she's one hard-working "problem solver."

Able to dig into your health care facility, study it carefully and emerge with new ideas about how you can use communications to increase efficiencies and contain costs.

For example, after thoroughly analyzing Methodist Hospital operations, Suzanne recommended a special feature Dimension 2000 telephone system.

The results — emergency room attendants receive vital information from doctors faster than ever, patients can easily contact admissions clerks handling their files and the hospital's telephone attendant requirements were cut in half.

To find out how a Southwestern Bell Account Executive can help you solve your health care problems and contain costs, call us today.

Southwestern Bell





A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC®

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

Wyeth Laboratories
Philadelphia, Pa. 19101

FOR MODERATE PAIN

A therapeutic dose of acetaminophen in *one* tablet

A therapeutic dose of two complementary analgesics

The convenience and economy of a dosage schedule of one tablet, every four hours as needed



WHY NOT WYGESIC® C IV

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbreviated Summery

INDICATION. For the relief of mild-to-moderate pain

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE. Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**)

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY. Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. (see **Warnings**) Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS. The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill, however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered, preferably IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

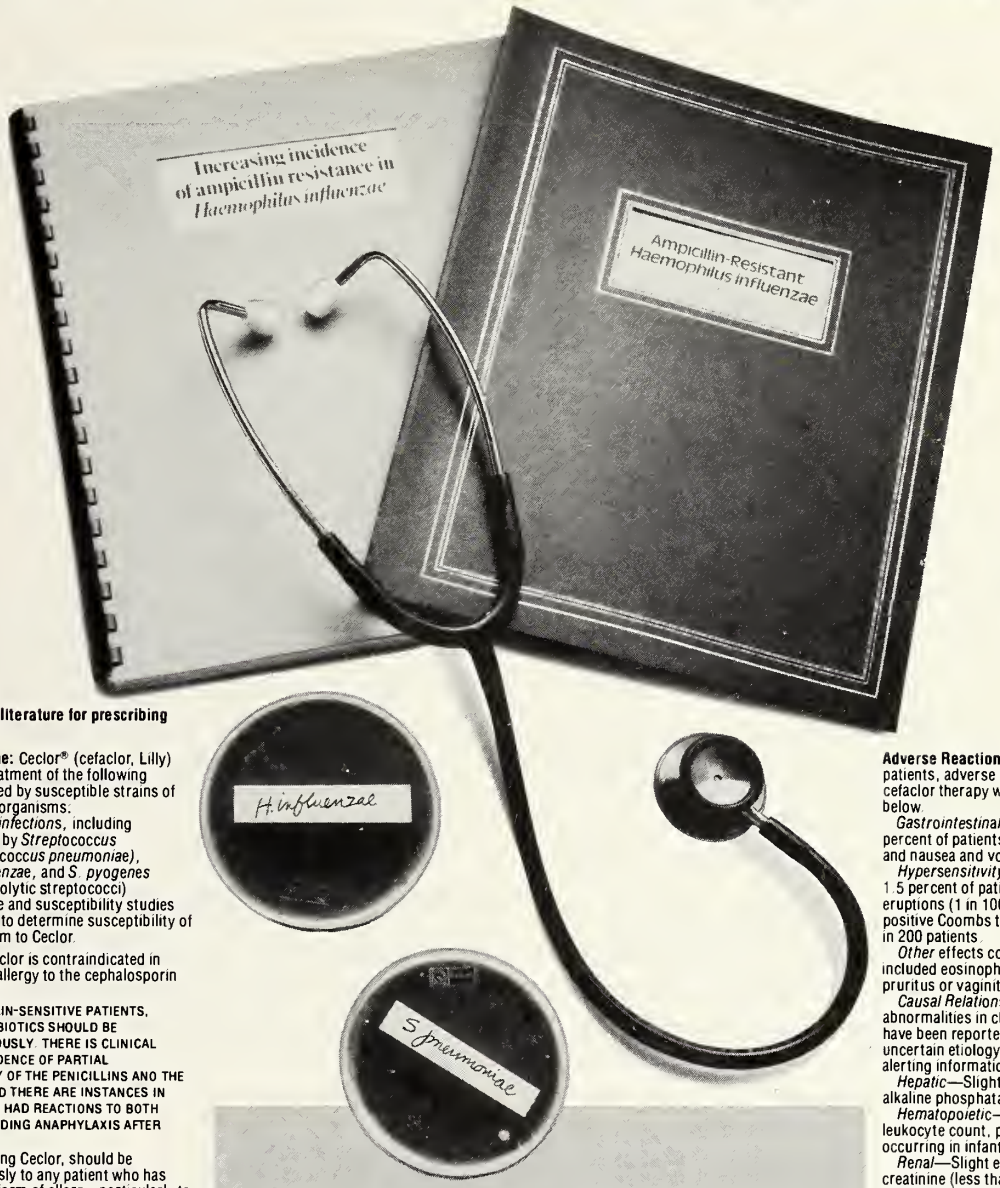
Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information. (JAMA 237:2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Wyeth Laboratories
Philadelphia, Pa 19101

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor® (cefaclor) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

1. Antimicrob. Agents Chemother., *8*:91, 1975.
2. Antimicrob. Agents Chemother., *11*:470, 1977.
3. Antimicrob. Agents Chemother., *13*:584, 1978.
4. Antimicrob. Agents Chemother., *12*:490, 1977.
5. Current Chemotherapy (edited by W. Siegenthaler and R. Luthy), II:880. Washington, D.C.: American Society for Microbiology, 1978.
6. Antimicrob. Agents Chemother., *13*:861, 1978.
7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

000482

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about 2.5 percent of patients and included diarrhea (1 in 70) and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in about 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less than 1 in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [070379R]

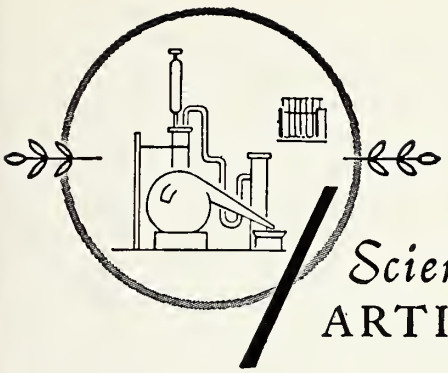
RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
George M. Penn, M.D., Topeka (913) 234-9566
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
Virginia L. Tucker, M.D., Lawrence(913) 843-3750
Kermit Wedel, M.D., Minneapolis (913) 392-2144

Kansas Medical Society, Topeka (913) 235-2383/235-3619



Antibiotics in Surgery

Current Concepts in Cepha Usage

RICK WINDLE, M.D.; GEORGE J. MASTIO, M.D., *and* GINGER FRENCH, Pharm. D., *Wichita*

RAPID DEVELOPMENTS in the field of antibiotic preparations make it increasingly difficult for surgeons to keep pace. Objective reviews have not been commonplace in surgical literature, forcing the surgeon to rely mostly on literature supplied by the pharmaceutical companies, which often precedes objective scientific data on antibiotic usage. The dilemma is reflected in recent statistics showing consistently low scores for surgeons on the National Antibiotic Therapy Test.[1] This study focuses on the cepha group of antibiotics, since 51 per cent of all antibiotics used by surgeons are from this group.

The cepha group represents more than 50 per cent of antibiotics administered by surgeons, and several new ones have been recently introduced. It is appropriate, therefore, to review the cephas and to focus on their individual properties.

Pharmacology

The common cephalosporins are cephalothin (Keflin), cephalexin (Keflex), cephradine (Velosef), cephazolin (Kefzol), cefamandole (Mandol), cefaclor (Ceclor), and the cephamycin, cefoxitin (Mefoxin). One that may soon appear on the American market is cefuroxine.

An excellent review of the properites of cephalosporins[2] states that with the exception of cefoxitin, the cephalosporins are semi-synthetic derivatives of cephalosporin C, an antibiotic substance obtained from the cephalosporium mold. Cefoxitin is more correctly a cephamycin rather than a cephalosporin, as it is derived from the streptomyces species. Referring to their chemical base, cephamycins are different from cephalosporins in that they have a methoxy group at the 7 position. Generalizing somewhat, it is the alterations of the seven amino positions that change antibacterial activity, while variance at the 3 position affects the drug's pharmacology *(Figure 1)*.

Most of the cephalosporins are susceptible in varying degrees to the enzymes that bacteria can elaborate to inactivate the antibiotics. These enzymes are known as the beta lactamases.[2] To review briefly, beta lactamases are plasmid-transmitted enzymes; a plasmid is a floating DNA fragment that is

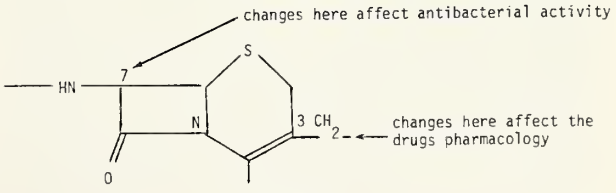


Figure 1

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capable of giving bacteria the genetic information to produce enzymes that will inactivate the antibiotic. What is of particular concern is that gram negative bacteria can pass many of the plasmids between different species. Multiple resistances can be mediated by a single plasmid. This was first demonstrated in Japan in 1959 when an isolate of *E. coli* resistant to four antibiotics transferred this same resistance to an isolate of *Shigella*. Fortunately, unlike the gram negative bacteria, the gram positive staphylococci plasmids usually mediate resistance to only one type of antibiotic. These plasmids do not appear transferable between different species. The implications of plasmid-mediated resistances are twofold. First, antibiotic resistance is tied directly to the amount of antibiotics used. The larger the volume and the variety used, the more the resistances are developed. Secondly, a compound that is more stable to an array of differing beta lactamases should in theory be a better antibiotic, as bacteria are less likely to destroy it. Of the antibiotics discussed here, cefoxitin is the most stable to degradation by these enzymes.

The antibacterial activity of cephas is directed at the cell wall killing the bacteria. Broadly addressing their spectrum of activity, cefoxitin and cefamandole may be less active against *Staph. aureus* than other cephalosporins. Many of the penicillin-resistant strains of *Staph.* are sensitive to cephalosporins. Most rare strains that are resistant to methicillin are also resistant to cephalosporins. Benzyl penicillin still is the preferred drug of choice against *staphylococcus,* coagulase negative. *Fecal streptococci,* Lansfield Group D, are resistant to all cephalosporins. *Pneumococci* are moderately sensitive with cefamandole being the most active. *Clostridium welchii* is sensitive to the cephalosporins but some of the other *Clostridiums* may be more resistant.

Against gram negative organisms these antibiotics have a wide range of activity. They are active against most clinical isolates of *E. coli, Klebsiella,* and *Proteus mirabilis*. Cefamandole and cefoxitin are generally much more useful against the gram negative bacteria than the older cephalosporins; they are more active against *Proteus* and *Serratia*. Cefoxitin is the only one that has shown a useful degree of activity against *Bacteroides fragilis*. Mandole appears to be the most active against *Hemophilus influenza*. Of the newer cephalosporins, cefuroxine and cefoxitin appear to be active against *Niesseria gonorrhea*.

Most cephalosporin is administered either intravenously or intramuscularly. The most easily tolerated intramuscular cephalosporin is cephazolin. Most of the others are painful on injection and need to be given intravenously. Cephalexin is the one most commonly used for oral consumption and is absorbed from the gastrointestinal tract.

A very important factor in cepha usage is consideration of the serum half life of the various cephas so that one can choose appropriate dosing intervals. A rough rule of thumb is that adequate serum levels of the antibiotic are maintained for approximately four times the half life. An example is cephalothin, which has a serum half life of 30-45 minutes. Appropriate dosing intervals to maintain an adequate serum concentration should be every two to three hours.

There is controversy about the importance of protein binding among the cephalosporins. If an antibiotic appears to be maintained within the vascular space because of high protein binding, this theoretically indicates that it will have poor penetration of the tissue. This means, in general, in the absence of inflammation or vasculitis, highly protein bound antibiotics have more difficulty reaching peripheral tissues. The exception appears to be cephazolin which is 75-85 per cent protein bound, but has tissue levels four times that of cephalothin which is approximately 10 per cent protein bound. Some people believe that protein binding is not of clinical importance until it exceeds 90 per cent.

All cephas are excreted primarily through the kidneys, mostly unchanged. The exception to this is cephalothin which is metabolized to a less active metabolite after one half life. Because of primary renal excretion, the dosages need to be adjusted for renal impairment. An estimation for adjustment is as follows:

$$\text{Dose} = \text{Normal Dose} \times \frac{\text{Patient's CrCl}}{\text{Normal CrCl}}$$

There is controversy about the cephas effect on renal function. Clinical evidence suggests that the use of cephaloridine, when used in conjunction with furosemide or ethacrynic acid, may be potentially nephrotoxic. Cephalothin has been suspected of renal damage when combined with gentamicin. It appears to be a synergistic relationship. Cephalothin has also been implicated in the development of an interstitial nephritis. The other cephas have not been implicated.

Additional properties of cephas are that they penetrate the meninges poorly and that they have low tissue concentration in the cerebrospinal fluid. If inflammation is present there is increased passage of the drug across the blood brain barrier, but these are rarely the drug of choice in any central nervous system infection. Many of the cephas have high biliary levels of concentration. Renal concentrations are expectedly high.

In addition to possible nephrotoxicity, administration of this drug is associated with pruritis and rash. A direct positive Coomb's test has accompanied the use of cephalosporins. Cephas taken by mouth may be accompanied by gastrointestinal upset. Phlebitis is associated with intravenous administration. They are associated with false positive Clinitests for glucose in the urine. Testape should be used.

Individual Properties

The main disadvantage with oral cephalexin is that it is far less active than the parenteral preparations. It has a poor degree of activity against staphylococci in general. Cefaclor is a new addition to the cepha market and may be taken orally. Its chief indication will be in treating ampicillin resistant *H. influenza,* especially in otitis media. It is also reasonably effective in controlling *staph, S. pneumoniae, E. coli,* and *N. gonorrhea.* Available dosages include 125 mg, 250 mg, and 500 mg, to be administered three times/day. Its disadvantage is expense, as it is four times more costly than amoxicillin.[3]

The two most commonly used cephalosporins are cephalothin and cephazolin. These two have similar antimicrobial spectrums working against *Staphylococci, Pneumococci* and *Streptococci* of the gram positive strains, exclusive of Enterococci. They are effective against many strains of *E. coli, Klebsiella,* and *Proteus,* but *Enterobacter, Serratia,* indole positive *Proteus, Providentia,* and *Pseudomonas* are resistant. They are also ineffective against *Bacteroides fragilis.* The usual minimum inhibitory concentration that will effectively eradicate bacteria is usually lower in the use of cephazolin than with cephalothin. Tissue levels are four times greater with cephazolin than with cephalothin. The half life of cephazolin is 90 minutes vs 30-45 minutes for cephalothin. Cephazolin may be administered intramuscularly. Cephalothin has been associated with sterile abscess and painful injection intramuscularly. Cephazolin may be given every six to eight hours; cephalothin needs to be given every two to four hours. Five hundred mg of cephazolin may be used where 1 gm of cephalothin may be sufficient. Up to 20 gms/day of cephalothin may be given in serious infections. Both seem to darken the urine. Cephazolin has high concentration in the bile, while cephalothin has relatively low concentrations. It is important to note that cephalothin, unlike most of the other cephalosporins, is rapidly metabolized to a less active substance. This metabolite is one fifth as potent against *Staph.* as its precursor.

Cephradine causes pain on intramuscular injection. It may be given intravenously or by mouth. The parenteral preparation has a high sodium content. It is generally less active than the other cephalosporins. At present it does not appear to have any advantages over other parenteral cephalosporins.

Cefuroxine, which may become available in the United States, appears to have good all-around properties. It is resistant to many gram negative beta lactamases and therefore has a wide spectrum. It is particularly active against *H. influenza* and *N. Gonorrhea,* including the beta lactamase-producing strains of both. It is not as active as many cephalosporins against *Staph. aureus.* Cefuroxine must be injected and would appear to be free from serious toxicity. The usual adult dose is 750-1500 mg three times/day.

Cefamandole has enhanced resistance to beta lactamase, but this resistance is not as pronounced as the other newer compounds, cefoxitin and cefuroxine. It has activity against *Enterobacter, indole-positive Proteus, Providentia,* and *Hemophilus influenza.* In general, it is better against most of the gram negative rods than cephazolin or cephalothin. It is less active than cephalothin or cephazolin against the gram positive species. In summary, cefamandole — as compared to the older cephalosporins — picks up many of the gram negative bacteria that cephazolin and cephalothin miss, but some gram positive activity is sacrificed. In addition, cefamandole also works against *Salmonella typhi.* It is not effective against *Bacteroides.* Its biliary concentration is 100 times greater than cephalothin and it is included in the enterohepatic circulation.

Cefamandole combined with aminoglycoside appears to be synergistic in its activity against *E. coli, K. pneumonia,* and *Enterobacter.* It may produce diarrhea. Its nephrotoxicity potentiation with aminoglycosides remains undetermined. It appears safe to administer this drug to patients with a history of penicillin allergy, excluding patients with previous anaphylaxis. Soft tissue and bone concentrations may be such that this medication would be useful in certain osteogenic infections. Clinically it appears this drug may be indicated for use in infections with mixed respiratory or bowel flora, urinary tract infections, orbital or facial cellulitis in children, and possibly some types of meningitis. The advantages of this medication are its broad spectrum, low toxicity, and high degree of toleration when given intravenously or intramuscularly. Usual adult dosage is 1 gm/six hrs.

Cefoxitin is the new type of antibiotic referred to as a cephamycin, and it has recently become available in the United States. It is expected that better cephamycins will be available in the future. This

antibiotic has several special qualities that make it particularly appealing. Cefoxitin appears stable to degradation by the beta lactamases. It has good serum, urine, and bile concentrations. Serum half life is 45-60 minutes. With the exception of *Pseudomonas aeruginosa,* cefoxitin is highly active in vitro against gram negative bacillae. This includes indole positive-producing *Proteus* species and *Serratia.* It is generally less active against gram positive organisms than the first generation cephalosporins. It is active against anaerobic organisms including *Bacteroides fragilis.* It may have potential for treating resistant *N. gonorrhea.* It is currently not approved for pediatric usage; it is of low toxicity; very few adverse reactions are reported. Some transient elevation of live enzymes and occasional phlebitis may accompany its usage. A few patients have experienced transient leukopenia and neutropenia as have been reported with other cephalosporins. False urine clinitests for glucose and false positive Coomb's tests have been noted. Appropriate dosing regimen would be 1-2 gms every four to six hrs. Of particular note is that there has been very little development of cefoxitin resistant isolates during the various periods of study included in most cumulative reports.

Until recently most physicians have not considered the cephalosporins to be the drug of first choice in the treatment of any active infection. Their broad spectrum would seem to make these agents choices for prophylaxis and for treating an infection until sensitivities return.

When these medications are used for prophylaxis, they should be in the serum prior to the start of surgery. This means either intramuscular injection prior to entering the operating room, or intravenous medication prior to the induction of anesthesia. A dosage may be given during surgery. Dosages should be continued for 24-48 hrs. At that point, the effectiveness of prophylaxis is not extended by continued administration.

Table I compares the relative costs in the Wichita area. Costs vary according to the bids accepted on individual drugs by each hospital. Each surgeon will be able to determine which antibiotic is preferable for use in selected instances.

Cepha usage for the initial treatment of infection pending the results of culture and sensitivity requires careful consideration. When the results of the culture are available, the patient should be placed on the appropriately sensitive drug of choice. Continuing patients on a broad spectrum antibiotic only contributes to increasing the number of nosocomial infections, which already account for an astounding 30-40 per cent of all hospital infections today.

It may be that cefamandole and cefoxitin, with their enhanced gram negative spectrum, may supplant other more toxic preparations — such as the aminoglycosides — when treating gram negative infections.

In conclusion, it appears that intelligent usage of the cepha antibiotics provide good prophylaxis for infections. They are occasionally the medications of first choice in treating infections and often the agents of second choice. The properties of reasonable cost, low toxicity, and broad spectrum make this antibiotic group an attractive choice.

References

1. Gardner, F. and Polk, H. C.: Further definition of antibiotic use and abuse in the surgical setting. *Arch. Surg.* 119:888, 1979.

2. Wise, Richard: Use of antibiotics-cephalosporins. *Br. Med. J.* 2:40-42, 1978.

3. *The Medical Letter.* 21:21, 1979.

TABLE I
APPROXIMATE ANTIBIOTIC COSTS TO THE PATIENT IN WICHITA*

	Amount	*Average Cost*	*Per Day in Recommended Dosage*
Cephalothin	1 g	$ 5.50	$33-$44
Cephazolin	500 mg	$ 6.50	$20-$26
Cefamandole	1 g	$ 9.00	$36-$54
Cefoxitin	1 g	$10.40	$62
Pen 6	5 mil units	$ 4.10	$25
Carbenicillin	5 g	$ 8.50	$51
Gentamicin	80 mg	$ 8.50	$25

* Excluding Costs of Administration

Fractured Fat

Traumatic Lipomata

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THE FATTY LAYER of tissue in the human body produces a gentle smooth roundness, converting irregularities to gentle contours. Of more importance is the cushioning effect it produces for deeper structures and the protective lining it gives to the skin. This is afforded by the sponge-like composition of the panniculus adiposis and its natural resilience. Changes in this layer of tissue causing abnormal fat deposition can occur in conjunction with endocrine or nutritional obesity, or as a localized mass of fat representing a true neoplasm. Pathologists have some difficulty in precisely defining the term "lipoma." It may, in some situations, simply represent a prolapse of normal fat through a disruption in the enveloping fascia by trauma to the local area. It is this group of traumatic lipomata that is the subject of this paper.

This condition has been reported by Meggitt & Wilson.[1] In their twelve cases, all patients were female and between the ages of 6 and 60 years. The lipomata were situated in the gluteal, trochanteric or lower lumber fat pads, and occurred following local blunt trauma due to direct impact from a car or from falling heavily on the area. This condition, though uncommon, is probably more frequent than is realized and is misdiagnosed as a resolving hematoma. An increased awareness of the pathology of this condition should result in more accurate treatment by correct diagnoses.

The following report of five cases of traumatic lipomata include two in males as well as one in the lower leg.

Case Reports

Case One: An 18-year-old white female was referred because of a large, soft swelling in the left posterior superior thigh. Twenty months previously she had been riding a bicycle when hit by a car. She sustained extensive bruising of the left posterior thigh which, upon absorption, left the large soft swelling. Three months after injury, partial excision of the residual swelling was carried out by an orthopedic surgeon; the pathologic report stated "consistant with a lipoma." Initial examination revealed a ten-centimeter transverse healed incision with a large depression above and a 9 x 12 cm soft non-tender protruding mass below.

At operation under general anesthesia through the old incision, a large fibro-fatty mass was noted to have formed, free from deep fascial attachment except at the location of the previous surgery. The subcutaneous tissue seemed to have been split with inferior retraction of the fatty tissue. Partial excision of the mass was carried out, but an advancement repair into the defect was not possible. Examination two years later revealed a residual bulge which decreased in size following considerable weight loss.

Abnormal fat deposition may be due to disruption of the enveloping fascia by trauma to the local area, and probably occurs more frequently than realized. An increased awareness of the condition could reduce the incidence of misdiagnoses and afford more appropriate treatment.

Case Two: A 28-year-old white female was initially seen because of a large soft tumor of the left lateral thigh. Seven months previously she was struck by a car and sustained blunt trauma to the lateral thigh; a large hematoma resulted without fracture or laceration. The hematoma gradually subsided without infection, but a swelling remained. Four months following the injury, through a 4 cm incision, exploration was carried out by an orthopedic surgeon for evacuation of what was felt to be a residual hematoma. The operative report states, "There was found to be . . . a hole where the hematoma had previously occupied. No gross blood was identified. There was a separation between the fat and fascial planes. . . ." Initial examination revealed a soft non-tender mass (10 x 8 x 2.5 cm) protruding just below the left greater trochanter and accompanied by a depression immediately above.

At operation under general anesthesia, a fibro-fatty mass was excised; a fascial cleavage plane

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Figure 1. Residual defect in *Case Two*. This is 3 years following injury and 2½ years after last surgery.

existed superiorly with downward displacement of normal fatty tissue into a bulging mass. Scarring from the previous operation prevented any accurate attempt at superior replacement advancement. The cosmetic appearance was considerably improved although a definite depression remained.

Case Three: An eight-year-old white female was seen at the mother's request for possible cosmetic improvement of a soft tissue swelling on the left lower leg. Five weeks previously she was involved in a skiing accident in which a ski rope became wrapped around her left leg. Emergency room records revealed ecchymosis and tenderness along with a rope burn across the lower leg. The tenderness subsided but swelling persisted in the area. Examination revealed a healed abrasion of the anterior lateral lower leg with a depression under and above the scar and a soft slightly tender mass (7.5 x 6 x 2 cm) below the scar and just above the ankle. This appeared typical of a fat fracture displacement and lipomatous mass below. Upon learning that its only significance was cosmetic, the parents declined surgical intervention. Re-examination two years later revealed that the mass was unchanged.



Figure 2. Soft swelling and depression at 3½ years following injury in *Case Three*.

Case Four: A 14-year-old white male was seen because of a depression of the left thigh and possible muscle problem. Three weeks previously he was standing behind a parked car and 15 feet from a second parked car. A third car, driven at high speed, rammed into the second car, throwing it forward into the first car and crushing the legs of the boy between the bumpers. Examination revealed a large depression in the medial posterior right thigh with a bulging soft mass (9 x 7 x 2 cm) immediately below. There was no real loss of muscle strength. Surgery was declined. There was no change at 18 months follow-up.

Case Five: A 22-year-old black male was standing in the street when a person in a car attempted to run over him. He was struck on the left side by the bumper before being dragged under the car. Beside an extensive facial avulsion, a large mass was noted in the left thigh. X-rays failed to reveal a fracture, and as time passed the large mass persisted. The impression of the plastic surgeon was a residual hematoma. No surgery was ever performed on the thigh. The patient presented three months following the injury seeking facial reconstruction. Examination at that time revealed a non-tender painless soft mass of the upper lateral left thigh. The bulging tumor measured 12 x 5½ x 1½ inches in size and had a definite depression immediately above it. There was no associated loss of muscle strength. He desired no surgical intervention.

Discussion

The panniculus adiposus is structured so as to maintain a resilient cushioning and distribute normal body loads over the body surface. Compression is possible by the fat lobules contained under pressure by a fibrous capsule and various septa. The fat-filled compartments are bounded by horizontal fibrous septa supported by vertical septa anchoring the epidermis to deep fascia. This is easily seen as fat lobules spring forth from their fibrous compartments during elective incisions in the buttocks and thigh regions. Larger blood vessels enter through the deep fascia and supply the subcutaneous tissue with a rich vascular network.

Meggitt describes the response of this layer to trauma very well in his article:

> Under normal body loads, local pressure produces flattening of the lobules and transference of the weight over a wider area, but the fibrous supporting septa prevent lateral migration of the fat. On release, the compression tension system enables the tissue to return to its resting shape and position. If sudden excessive force is applied locally, such as impact from a moving vehicle or heavy falling on the area, the fat compartments are burst open with rupture of



Figure 3. Deformity at 15 months following injury and no surgery in *Case Five.*

the septa and shearing off of the anchorage between the skin and the deep fascia. Also, with local vascular damage, a varying amount of bruising and hematoma formation occurs. . . .[1]

Formation of a traumatic lipoma results from the "fat fracture" by the opening of fascial planes and retraction of the detached subcutaneous tissue into the adjacent body area. Absorption of the hematoma and bruising produces the typical deformity of a soft tissue defect above and a soft protruberance below. We have indeed learned from the mammoplasty and abdominal lipectomy that fatty tissue characteristically does not undergo local resorption or regeneration, thereby making this a permanent deformity in adults.

All previously reported cases were in females, but it can also occur in males, as shown in cases four and five. Since the deformity is cosmetic rather than functional, it may be more disturbing and correction more readily sought by the female. Pain and tenderness were not major factors in causing these patients to seek help. No mention was made of race in the previous study, but this probably is not a factor.

Treatment of a traumatic lipoma may be by excision, thereby retaining the depression already present while removing the bulge. This is recommended when pain or tenderness is the problem. Surgical repair by advancing the fibro-fatty mass into the defect is also a possibility, although usually difficult or impossible, especially following previous exploration as in cases one and two, but should give the best result.

Summary

Traumatic lipoma formation was discussed as to its history and pathologic findings. Five new cases were presented, including for the first time both males and a lower leg deformity. Treatment of the essentially cosmetic deformity was discussed.

Reference

1. Meggitt, B. F. and Wilson, J. N.: The battered buttock syndrome — fat fractures. Br. J. Surg. 59:165-169, 1972.

Human Protein Requirements

(Continued from page 266)

condition in the United States. It is also known that the quality of protein consumed has changed since 1900.[12] More than two-thirds of dietary protein comes from animal sources today, as opposed to one-half seventy years ago, which reflects a greater grain consumption at that time.[12] In terms of achieving a high-fiber, low-fat diet which has been suggested by some health professionals for improved health, a shift toward increased consumption of grain and vegetable proteins is desirable.

Since some elderly individuals suffer from various socio-economic and disease conditions that may lead to poor eating habits, special consideration has been given to the adequacy of their diet in terms of the major nutrients. The Ten-State Nutrition Survey reported that while total nutrient consumption among the elderly tended to be below the 1968 RDA, protein intakes met or exceeded the RDA in the majority of instances.[7] Furthermore, recent metabolic studies on protein requirements in the elderly concluded that a typical American diet with its high protein content should be adequate for this age group.[13] Certain elderly ethnic groups, e.g., black and Spanish-American women in high income states, had mean protein intake levels below the RDA. This observation, however, does not necessarily signify the existence of protein malnutrition among these people, since RDA are set well above minimum requirements.

An evaluation of the protein intakes of various groups of vegetarians demonstrated that "pure" vegetarians (vegans) were receiving about 83 grams of dietary protein/day while the lacto-ovo vegetarians (i.e., including milk and eggs) consumed about 98 g/day.[14] A more recent report confirmed that vegetarian diets contain adequate protein levels when the diet is selected from a wide variety of vegetables, grains, nuts, and fruits.[7]

Protein Needs and Weight Reduction

Information is also available on the protein needs of subjects who are on strict weight reduction regimens which require fasting or starvation. Without an exogenous source of calories the body must use its endogenous reserves of fat and protein primarily to meet energy requirements. In short-term starvation the body adapts to the caloric deprivation by using muscle protein and adipocyte fat as its primary source of calories. In long-term starvation (longer than 2 weeks), the body begins conserving protein and calories are derived primarily from fat.[15] Protein losses are continuous, however, and replenishment is vital to ensure survival. Obesity therapy regimens which use long-term unsupplemented fasting may jeopardize the subject's health through excessive loss of functional tissue proteins, minerals, and vitamins. Supplemental fasting modified by administration of dietary protein, i.e., "protein-sparing therapy,"[16] in part enables the fasting subject to

(Continued on page 261)

Splenectomy for Trauma

A Community Hospital Experience

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THE SPLEEN'S relationship to other organs in the left upper quadrant renders it vulnerable to injury from externally applied forces, penetrating wounds, or inadvertent injury during other surgical procedures. The current emphasis on splenic preservation to minimize post-splenectomy sequelae encourages surgeons to protect the spleen when possible. The use of debridement and partial amputation or ligation techniques and topical hemostatic agents has resulted in fewer splenectomies. In the occasional patient in which removal of a severely traumatized spleen is unavoidable, the introduction of pneumococcal vaccine (Pneumovax) has afforded splenectomized patients some protection from septic complications of splenic absence.

Methods and Results

All splenectomies performed at Wesley Medical Center between 1974 and 1978 were retrospectively reviewed. Splenectomies for hematologic disease were excluded. Included in the study were all cases of splenectomy during another surgical procedure, and blunt or penetrating abdominal injury with splenic involvement *(Table I)*.

The splenectomies were evaluated as to type of associated surgical procedure, concomitant injuries, and mortality. The value of a spleen scan was also reviewed in the equivocal patient with blunt trauma.

The age range in this series was from one day to 87 yrs. with an average age of 32 yrs. Motor vehicular accidents accounted for 41 per cent of the injuries, while iatrogenic injuries were involved in 39 per cent of cases. Thirteen patients were injured from a fall, a kick, athletic endeavors, or some other unusual circumstance. Three patients in the study had alleged spontaneous rupture of the spleen without prior trauma history, one occurring in the immediate post-partum period. Two patients sustained gunshot wounds, necessitating splenectomy, and a single case was from birth trauma. Three patients had ruptured diaphragms, and seven patients had associated liver injuries.

Iatrogenic splenic trauma involved a variety of surgical procedures *(Table II)*, usually confined to the upper abdomen. The most common of these was vagotomy with gastric resection or pyloroplasty in nine patients. Of this group, four had simultaneous hiatal herniorrhaphy. Four patients had a Nissen fundoplication alone, and one splenectomy was done during transthoracic hiatal herniorrhaphy. Interestingly, four procedures were incidental to vascular reconstruction, abdominal aneurysmectomy, or aortoiliac bypass graft. Five inadvertent spleen injuries occurred during cholecystectomy, three with simultaneous hiatal herniorrhaphy. Three left colectomies and one subphrenic abscess drainage resulted in splenectomy. One splenectomy was also performed during an exploratory laparotomy with adhesiolysis, and one during a transcolonic polypectomy at a splenic flexure.

The spleen is vulnerable to trauma. A retrospective review of 100 cases of splenectomy at Wesley Medical Center confirms the morbidity and mortality associated with splenectomy. Greater emphasis should be placed on splenic protection, and a more conservative approach to splenic injuries is advocated.

TABLE I

Mechanism of Injury	*No. Patients*
Iatrogenic	39
Auto accident	32
Fall, kick	13
Motorcycle	9
Spontaneous rupture	3
Gunshot wound	2
Birth trauma	1
Total	100

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TABLE II
PROCEDURES WITH WHICH INCIDENTAL SPLENECTOMY WAS ASSOCIATED

Vagotomy, antrectomy, Billroth II	5
Nissen fundoplication	4
Vagotomy, Billroth I	3
Vagotomy, pyloroplasty, hiatal herniorrhaphy	3
Vagotomy, Billroth II, hiatal herniorrhaphy	1
Vagotomy, pyloroplasty	2
Cholecystectomy, hiatal herniorrhaphy	3
Cholecystectomy alone	2
Gastric resection	3
Gastrojejunostomy	1
Left colectomy	3
Abdominal aneurysm	2
Aortoiliac bypass	1
Cholecystectomy, lysis of adhesions	1
Exploratory laparotomy	1
Gastrectomy for ZE	1
Drainage left subphrenic abscess	1
Transcolonic polypectomy	1
Transthoracic hiatal herniorrhaphy	1
Total	39

Associated injuries in major trauma patients included eight patients with rib fractures, six with pneumothorax or hemothorax, one skull fracture, one pelvic fracture, and six extremity fractures. Seven patients had associated liver injuries, and three had diaphragmatic ruptures.

The series included ten mortalities — three associated with surgical trauma while the rest succumbed to multiple blunt or penetrating injuries.

The value of the spleen scan in the equivocal patient with blunt trauma was also reviewed. Twenty patients had a spleen scan prior to surgery. One scan showed increased radiotracer uptake and was repeated. At surgery the specimen had a subcapsular hematoma. Three false negative scans were confirmed at the time of surgery.

Discussion

The nonoperative approach to splenic laceration or subcapsular hematoma has been advocated in the literature in recent years. Ein, in 1978, reported a series of 56 pediatric patients with proven damaged spleens.[1] In his series, 35 patients were successfully managed nonoperatively. He stresses restricted post-hospital activities for at least two months to avoid further aggravation of the injury.

As an alternative to surgical intervention, the scintiscan is a useful means of nonoperative management. In 1978, Howman-Giles reported a series of 28 patients managed by serial 99m Technetium-sulfide colloid scintigraphy, confirming splenic disruption.[2] This method is noninvasive and radiation exposure is minimal. He stresses that this modality of treatment is feasible provided the patient's condition is stable, and intravenous fluids maintain that stability. Evidence of clinical deterioration warrants surgical intervention. Conservative management involved three weeks of bed rest with followup scan before discharge.

In 1976, Grosfeld *et al.*[3] studied the effects of splenic trauma in animals. They compared mortality from splenic trauma without therapy to splenectomy, hemisplenectomy, and primary spleen repair. A zero mortality rate was noted following the three surgical techniques. This study confirms the concept that splenic salvage is a feasible alternative to splenectomy.

An alternative to hemisplenectomy has been the use of topical hemostatic agents such as Avitene. This was studied in 1974 by Morgenstern in dogs. After 12 weeks, tissue reaction was minimal and all traces of Avitene were gone.[4]

In the past, subtle splenic injuries presented a diagnostic dilemma, and days or weeks later secondary hemorrhage occurred following the latent period of Baudet.[5] In actuality, Olsen noted in 1977 that the sequelae were from a delay in diagnosis rather than a late rupture.

The results of this study would suggest a high percentage of iatrogenic splenic injuries and resultant splenectomy for trivial lacerations. Simmons reviewed complications of transabdominal vagotomy and reported splenic trauma as the most frequent complication.[6] Eighteen of his twenty cases resulted in splenectomy. Wagensteen described a technique of gastric mobilization prior to vagotomy to minimize splenic trauma.[7] He advocates opening the gastrohepatic omentum and dividing vessels along the greater and lesser curvatures of the stomach for greater exposure.

Morgenstern calls the resultant splenectomy from operative injury accidental splenectomy and believes it accounts for 25 per cent of all spleens removed. He stresses the knowledge of peritoneal anatomy of the spleen with prophylactic division of these folds where feasible to lessen capsular avulsion injuries.[8]

The post-splenectomy patient is at high risk for developing overwhelming post-splenectomy sepsis syndrome. A mortality of 50 per cent from post-splenectomy infections can be expected in the first two years of life.[3] Although the incidence of sepsis is relatively low, the fatality rate is exceedingly high.[9] Gopul in 1977 reported fulminant infection in five

asplenic patients without underlying disease who succumbed to infection.[10] Diagnosis was established by culturing abundant pneumococci in blood. Current literature suggests pneumococcal vaccine (Pneumovax) for any post-splenectomy patient. However, polyvalent polysaccharide vaccine cannot be guaranteed to protect each patient from each type of pneumococcus for which the vaccine is designed. It is not recommended currently for the pregnant patient and is not advocated in the child less than two years old.[8]

Conclusion

Results of this review would suggest that incidental splenectomy is commonly associated with procedures in the upper abdomen. This compares favorably to Danforth's report of 981 consecutive splenectomies which showed 20.5 per cent involved gastrectomy for peptic ulcer disease while 9.7 per cent accompanied hiatal herniorrhaphy.[11] This review suggests that multiple simultaneous upper abdominal procedures increase the likelihood of splenic damage. However, our series of 39 per cent iatrogenic injuries is considerably higher than the usual 25 per cent quoted in the literature.

Review of the literature supports our contention that in most cases after blunt or penetrating trauma to the spleen, conservative measures are feasible; such measures include topical hemostasis, partial splenectomy, or omental patching with suture repair.

The spleen lends itself to injury from a variety of mechanisms. A more conservative approach at surgery or nonoperative treatment is advocated for spleen preservation, and the spleen scan is valuable in following these patients. Perhaps unilateral surgery will decrease the incidence of iatrogenic injury. A more concerted effort toward splenic protection should be adopted to obviate post-splenectomy complications.

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NATURAL IMMUNITY

Who says women are the weaker sex? Two physicians writing in the American Medical Association's *Journal of Diseases of Children* present evidence that females are naturally immune to certain diseases.

David T. Purtilo, M.D. and John L. Sullivan, M.D., write that "Evolutionary selection has equipped females with immunoregulatory genes on the X chromosome for coping with life-threatening illnesses. Females, who have one more X chromosome than males, are less likely to get some infectious diseases and certain forms of cancer."

Through the life cycle, males show a decreased survival rate. Although there are five per cent more male babies born than females, that number diminishes until, in later life, women outlive men by eight years. Some explanation for this mortality rate lies in environmental exposure.

"In addition to accidents, severe infectious diseases are responsible for many deaths in males. Severe respiratory infections with parainfluenza show male preponderance."

Studies show that males experience more staph infections and, during recent epidemics of Legionnaires' disease, three times more men than women fell victim to it.

There are genes on the X chromosome that make females more immune to these diseases. By having *two* X chromosomes, females have a natural survival advantage over males.

Wound Closure

The Use of Musculocutaneous Flaps

GEORGE L. PELTIER, M.D.; MANI M. MANI, M.D.; *and*
DAVID W. ROBINSON, M.D., *Kansas City, Kansas*

The use of musculocutaneous flaps has evolved into an effective surgical technique for wound closure. The versatility of this procedure is demonstrated in this review and discussion.

Reconstructive surgeons, during the past fifteen years, have investigated the use of muscle to cover the loss of skin over critical tissue. These areas include the pretibial skin, chest wall, abdominal wall, major vessels, pressure-bearing areas, and tendons. Methods of wound closure have evolved from simple muscle flaps to composite musculocutaneous flaps. The muscle in these flaps acts as a carrier for the cutaneous segment of the flap. The skin has its vascular supply from the underlying muscle and the composite flap is turned on the arc of the dominant vascular pedicle of the muscle. Three cases in which these flaps were used are presented and discussed.

Case One: A 49-year-old female underwent a right modified radical mastectomy for carcinoma of the breast. This patient's axilla contained no positive lymph nodes, and she had no evidence of recurrence. Her skin wound healed well, but the skin and fat covering her chest wall were thin. She requested reconstruction of her breast for cosmetic reasons *(Figure 1).*

Due to her large ptotic left breast, left subcutaneous mastectomy was performed with a free-nipple graft. At the same time, a latissimus dorsi flap was rotated over her right chest for soft tissue coverage. The operative procedure consisted of a transverse incision within the bra line across the mid back. The latissimus dorsi was mobilized from its



Figure 1. Right modified radical mastectomy.



Figure 2. Latissimus dorsi breast reconstruction. The cross lateral area identifies undermined skin filled out with muscle.

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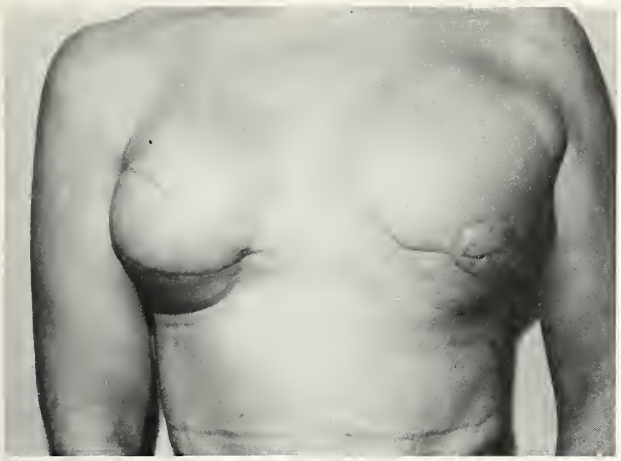


Figure 3A. Anterior view of bilateral breast reconstruction. The right breast was reconstructed with a latissimus dorsi flap and a silicone implant. The left was reconstructed with a submuscular implant.



Figure 3B. Lateral view.

origin in the low back. The flap was then turned onto the right anterior chest wall through an axillary tunnel. The insertion and the vascular pedicle of the latissimus dorsi remained intact. On the muscle flap, a segment of skin (measuring 5 x 8 cm) was carried. The donor defect was closed primarily under very slight tension. The skin of the anterior chest was incised and mobilized in the subcutaneous plane. The muscle with its overlying skin was then secured to the subcutaneous tissue and the rib cage over the anterior chest wall and the wound was closed incorporating the oval-shaped segment of skin *(Figure 2)*. Her postoperative course was uneventful except for a seroma on the low back, which resolved promptly with needle aspiration. Placement of bilateral silicone implants was completed after her wounds had satisfactorily healed *(Figure 3 A and B)*.

Case Two: A 44-year-old male was burned by gasoline flame over his left anterior tibia. This burned skin was excised and grafted shortly after his initial injury. The wound healed promptly but remained unstable for 28 years. The area broke down easily with minor trauma and the tibia was frequently exposed *(Figure 4)*. The patient was taken to surgery and all necrotic and grafted skin was removed. Then, a medial gastrocnemius musculocutaneous flap was raised on its vascular supply and turned over the left anterior tibia. The posterior donor area was closed with split-thickness skin graft. The distal tendinous attachments of the muscle were divided sharply, and the gastrocnemius muscle was lifted from the soleus muscle. The vascular supply consisting of the sural arteries was carefully preserved. A small skin bridge remained intact. This large flap was then sutured over the anterior tibia providing composite muscle and skin coverage. The wound healed promptly and he was discharged 15 days following surgery. His gait was excellent and he returned to work soon after surgery with a Jobst stocking for support. He has had no further break down over his left anterior tibia *(Figure 5A-D)*.

Case Three: A 45-year-old male was critically injured while under a truck attempting to release an electrical brake when the truck rolled forward. His chest was pinned between a retaining wall and a tire causing a fracture dislocation of T12 and L1 with immediate paraplegia. Open reduction of his spine demonstrated a complete cord transsection. During the past ten years, he has had multiple admissions for genitourinary infections and decubitus ulcers of the heel, iliac crest, sacrum, and ischium. Eight years following injury, a local flap was rotated to cover a sacral decubitus. Because of continued superficial wound infection, bilateral gluteus maximus muscu-

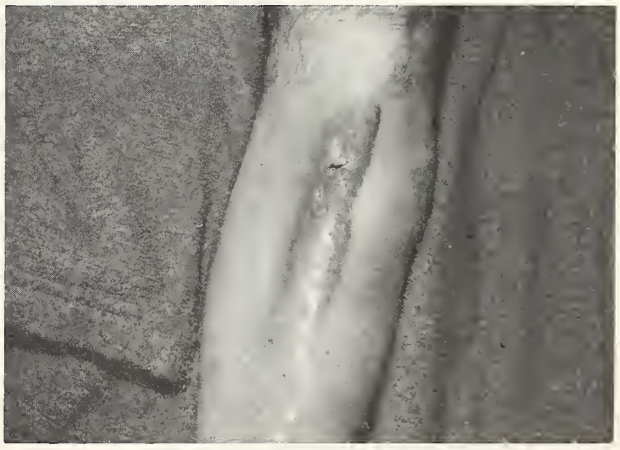


Figure 4. Unstable pretibial scar.



Figure 5A. Medial view of gastrocnemius musculocutaneous flap.



Figure 5B. Anterior view gastrocnemius musculocutaneous flap.



Figure 5C. Posterior view.



Figure 5D. Lateral view.

locutaneous flaps were turned for secondary wound closure two months later. The donor area was closed with a split-thickness skin graft. He then promptly developed an ulcer over his right ischium. This was debrided and closed with a gracilis musculocutaneous flap. The patient was operated on under general anesthesia and in the lithotomy position. The ulcer was debrided and an ischiectomy was performed. The soft tissue was then closed over the bony stumps and a right gracilis muscle with overlying skin was elevated and rotated into the defect. A circle of skin measuring 4 x 4 cm adequately filled the defect and the skin was closed primarily. A small area was regrafted because of a partial necrosis of some of the overlying skin. His wound healed slowly but with no further complications.

Discussion

Ralph Ger in 1960 reported his investigations into the use of the muscle flaps covered with a split-thickness skin graft for defects of the pretibial area.[1] He used muscles from the foot to cover ulcers over the ankle. In 1972, Orticochea in Spain reported the use of gracilis musculocutaneous flap as a cross leg flap to cover large traumatic lower extremity injuries.[2] In 1977, John McCraw and others reported experimental cadaver studies which clearly identify the blood supply to the skin from underlying muscles.[3]

Later, McCraw reported the use of latissimus dorsi flap for chest wall reconstruction.[4] This flap carries the latissimus muscle and its overlying skin based on a subscapular artery and the thoracodorsal artery. This large unit may be rotated up into the neck and also across the chest wall. The most common use for this flap is breast reconstruction. Arteriography may be needed to verify the patency of the thoracodorsal artery if this artery may have been ligated during mastectomy. If the thoracodorsal nerve, as demonstrated by lack of voluntary contracture, is not intact, the artery is very likely to have been divided. The resulting loss of strength from moving the muscle has proven to be minimal and should be taken into consideration only for patients engaged in strenuous activities requiring climbing, backward extension of the upper extremity, and adduction of the shoulder.

McCraw has also reported the use of the gastrocnemius musculocutaneous flap for closure of lower extremity wounds.[5] This flap provides an alternative to the cross leg pedicle flap that requires multiple procedures and long hospitalization. The medial head gastrocnemius flap may be used to cover the middle and upper tibia, the popliteal fossa, and 15 cm above the patella. The lateral head may cover the upper anterior and lateral shin but is restricted somewhat by the small area of skin that can be moved. Fluoroscein in vivo staining is recommended to demonstrate perfusion and, therefore, viability of this flap. The blood supply for the gastrocnemius flap enters the muscle through the sural arteries. This flap can be used for defects from osteomyelitis, pressure sores, open fractures, and thermal injury.

The gracilis myocutaneous flap, described by McCraw, can be used for reconstruction of the penis, vagina, and pelvic defects.[3] This flap will cover sacral and pelvic defects based on the vascular pedicle 10 cm below the pubic tubercle. The donor defect may be closed primarily and gives negligible disfigurement in all patients.

Conclusion

The musculocutaneous flap can be used for coverage of pelvic, chest, and lower extremity wounds. The use of these flaps avoids delaying procedures. The resulting muscle loss is easily assumed by adjacent musculature. Musculocutaneous flaps are extremely important procedures for closing large, soft tissue defects.

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Abdominal Aortic Aneurysms

Management and Avoidance of Pitfalls in Community Hospitals

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SURGICAL MANAGEMENT of abdominal aortic aneurysms is well established.[1-4] In many community hospitals, abdominal aortic aneurysms are handled by general surgeons with variable experience in vascular surgery. Problems with this surgical procedure in community hospitals are reviewed. Emphasis on management and avoidance of pitfalls are discussed as an aid to the practicing surgeon who may not see a large number of this type of case.

Materials and Methods

Charts were reviewed retrospectively for a three-year period (1976-78) for all abdominal aortic aneurysms managed on the senior author's service. A consulting or referring internist was involved in each case. Aortography was infrequently required. The surgical technique used has been discussed in vascular textbooks, and some of the fine points will be discussed in this paper.

Results

A total of 143 cases were reviewed. There were 117 males in the series (82%), and average age was 68 years. A high incidence of associated hypertension and arteriosclerotic cardiovascular disease was seen, and most patients smoked cigarettes. Seven patients had previously undergone coronary artery bypass surgery, and four underwent carotid endarterectomy prior to the abdominal aortic aneurysmectomy. One patient had undergone previous abdominal aortic aneurysmectomy in another city and presented with a false aneurysm four years later. One patient had disseminated intravascular coagulation from an intraluminal aneurysm clot which required preoperative treatment. Four patients (2.8%) had intra-abdominal malignancy discovered at laparotomy for aneurysmectomy. Eleven patients (7.7%) presented with a history of claudication, and one had undergone prior embolectomy.

Fifteen patients (10.5%) had ruptured aneurysms with free blood in the peritoneal cavity or a retroperitoneal hematoma discovered at the time of surgery. Thirteen of these patients were admitted in shock. Seven patients (47%) died, all within 24 hours of operation, and six deaths occurred on the operating table. The eight surviving patients all developed complications; seven had renal failure with hemodialysis required in six of these, and one had a prolonged ileus.

A series of patients with abdominal aortic aneurysms is presented. The operative mortality rate for elective, non-ruptured aneurysms (3.1%) and zero incidence of renal failure requiring dialysis in these cases form the basis for the discussion. Surgical management and avoidance of pitfalls in community hospitals are reviewed.

Overall mortality was 11 patients (7.7%), with an elective, non-ruptured mortality rate of four of 128 patients (3.1%). The non-ruptured mortality was caused by myocardial infarction in two patients, and cerebrovascular accident in two patients. Postoperative hospital stay averaged 13.7 days (range 6-28 days) for elective non-ruptured cases.

Data was then evaluated for 128 cases, excluding mortalities and ruptured aneurysms. A tube graft was used in 68 patients (55%). Bifurcation grafts were required to the iliac arteries in 32 patients (26%) and to the femoral artery (groin) in 23 patients (19%). Twenty-three patients (19%) had concommitant procedures performed. Nineteen of these were cholecystectomies, two splenectomies (one for disease and one iatrogenic injury), one vagotomy and pyloroplasty, and one renal artery bypass. Associated aneurysms were seen in 21 patients (17%). The majority of these were iliac (15) or femoral (4). Two patients had prior aortic arch or descending thoracic aortic grafts.

Clamping above the renal arteries was required in three patients, and none required dialysis for renal

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TABLE I
SPECIFIC COMPLICATIONS AND ADDITIONAL OPERATIONS

Complications	*# of Patients*
Prolonged ileus (> 10 days)	12
Severe atelectasis Pulmonary insufficiency Pneumonia	10
Peripheral emboli	7
Renal insufficiency (not requiring dialysis)	4
Myocardial infarction	4
Cerebrovascular accident	3
Congestive heart failure	3
Rectal bleeding (ischemic colitis)	2
Pleural effusion	1
Additional Operation	
Postoperative bleeding	3
Embolectomy	2
Above knee amputation	1
Toe amputation	4

TABLE II
SUMMARY OF DATA ON SMALL AND LARGE ABDOMINAL AND AORTIC ANEURYSMS

Large Aneurysms (> 6 cm. diameter)

1. Risk of rupture 45-50% (all cases observed over ten year period)
2. Operative mortality 5-10% (depends on associated risk factors)
3. Five year survival —
 with resection 50%
 without resection 5-10%

Small Aneurysms (< 6 cm. diameter)

1. Risk of rupture 15-20%
2. Operative mortality — 3% (patients free of severe associated disease)
3. Five year survival (comparable good risk patients)
 with resection 65-70%
 without resection 50%

failure. Mild azotemia was noted in four other patients, none of whom required dialysis. Seven of eight surviving patients with ruptured aneurysms required hemodialysis.

Complications were seen in 37 patients (30%), and eight of these patients had more than one complication. Specific complications and additional operations are listed in *Table I*. Most complications were due to associated systemic disease, and complications from technical errors were few. Five patients with peripheral emboli required amputations with four of these limited to the toes. One patient developed a pleural effusion from perforation of the internal jugular vein by a catheter and intrapleural infusion of fluid. Three patients were re-explored for postoperative bleeding; one patient had a splenic capsule tear, and two patients had large mesenteric or retroperitoneal hematomas.

Discussion

The primary purpose of this paper is to discuss surgical techniques in the avoidance of problems associated with abdominal aortic aneurysms. However, brief mention will be made in avoidance of pitfalls in other areas.

Aortography is not required in most abdominal aortic aneurysms (AAA).[5, 6] Sonography is a useful, simple non-invasive test to diagnose AAA, to assess its size, and to follow a small asymptomatic aneurysm or an aneurysm in a high surgical risk patient. Aortography is not useful to assess size of the aneurysm, since many times only a normal lumen will be seen because a laminated clot fills the aneurysm. Aortography may be used to evaluate the renal or visceral arteries when involvement of these vessels is suspected on clinical examination. Evaluation of concomitant severe aorto-iliac or femoral-popliteal disease is another indication. Translumbar or transfemoral aortography should not be used when AAA is suspected.

AAA is an expression of generalized or systemic arteriosclerosis.[1-3, 7] Many of the complications seen with abdominal aortic surgery are a result of this systemic disease. Renal function should be optimal since this may be adversely affected by the operation. *Table II* summarizes data on large and small aneurysms so that good surgical judgement can be used in these patients.[1, 2]

Modern grafts are a significant advance over the homografts initially used.[4] Long term failure rate is in the range of 2 per cent. The Dacron (polyester) microvelour knitted graft is preferable since it promotes better pseudointima formation and minimizes thrombosis. Silk suture should never be used in anastomotic sites since this may result in late suture line disruption and pseudoaneurysm formation. Tube grafts can be used in the majority of cases.

Proximal and distal control of the aorta is best performed without complete mobilization of vessels. Complete encirclement is not necessary in all cases for placement of the vascular clamps *(Figure 1)*. Attempting to place an umbilical tape circumferentially about the proximal aorta may result in



Figure 1. Proximal and distal control of the aneurysm are best performed without complete encirclement of the proximal aorta and iliac arteries. Adequate control can be obtained by clamping the lateral walls as shown. Passing an umbilical tape circumferentially (demonstrated on one iliac artery) may lead to dangerous bleeding.

rupture into the posterior wall or a torn lumbar artery. These are extremely difficult to control. Routine circumferential mobilization of the iliac arteries may tear into an iliac vein, resulting in bleeding that can be as troublesome as major arterial bleeding. Similarly, mobilization of an iliac aneurysm may result in severe pelvic bleeding. In these cases, the internal iliac ostium may be controlled by suture ligation from within the aneurysm.

The left renal vein may be ligated as it crosses the proximal aorta to gain additional exposure. This ligation should be performed close to the inferior vena cava to preserve collateral adrenal and gonadal venous pathways. Temporary clamping of the suprarenal aorta at the diaphragmatic hiatus through the lesser sac is necessary to control bleeding during dissection of an inflammatory or ruptured aneurysm. Minimal mobilization of the duodenum will decrease the incidence of injury to this bowel.

After blood is drawn for preclotting of the graft, systemic heparinization is performed prior to aortic cross-clamping. After cross-clamping the aorta, the aneurysm is incised and opened, and the thrombus removed. In order to avoid dangerous lumbar bleeding, the wall should not be resected. The lumbar vessels can be controlled by suture ligation in the sac. The wall of the aneurysm is viable, and there is almost no risk of infection in leaving the wall intact. Aorto-duodenal fistula is not limited to the proximal anastomosis, so the wall of the aneurysm sac can be used to cover the graft and exclude contact of the duodenum with the entire graft. Only if infection of the aneurysm is already present should resection be considered.[4, 10]

Diagnosis of a ruptured aneurysm demands an immediate operation.[8, 9, 11] Incision should not be delayed if the patient is in shock. Occasionally prep and draping before induction of anesthesia allows better control of the blood pressure. Proximal control of the aorta must be gained before dissection and opening of the retroperitoneum. This can almost always be performed through the lesser sac. Some surgeons advocate opening the chest first in ruptured AAA.[8] This allows for rapid proximal control of the aorta, and open cardiac massage may be performed if necessary. However, rapid proximal control can be obtained through the lesser sac in most cases. Exceptions would be a patient in profound shock, an obese patient, or where a high aneurysm is suspected.

Heparinization does not need to be performed in ruptured aneurysms since many of these patients receive large amounts of blood transfusions and generally develop clotting problems. For this reason, a non-porous, woven graft should be considered in these cases. To facilitate surgical repair, a polypropylene suture may be used to place posterior wall sutures in a continuous fashion without the graft and aorta in close apposition *(Figure 2)*. This suture may then be easily pulled taut, facilitating the anastomosis. As can be seen in our series as well as other series,[8, 9, 11] the mortality rate for ruptured AAA remains high.

Anatomic variations, such as multiple renal vessels, must always be kept in mind. Accessory renal vessels should be re-implanted into the graft with a button of tissue. Infrequently, other anatomic variations are encountered, such as the horseshoe kidney. Division of the isthmus is rarely required, although blood supply to the isthmus can be ligated with impunity to gain further exposure.

Avoidance of Complications

A variety of complications inherent to AAA surgery can be avoided with careful attention to technique.[10] Thrombosis of the graft may occur from pseudointimal flap, infection, poor runoff, or kink-

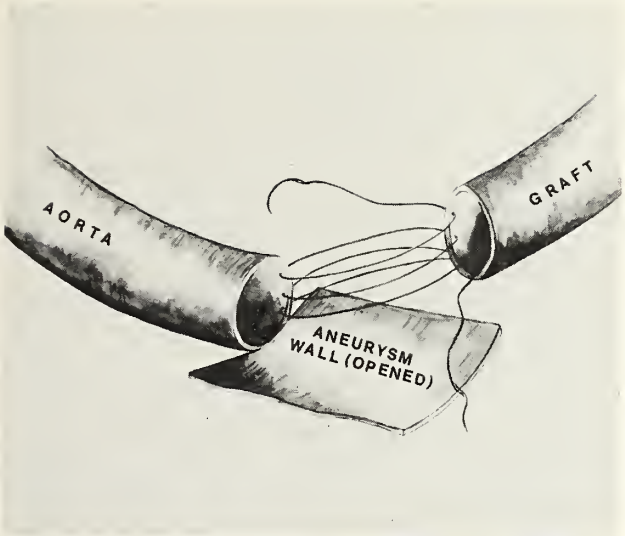


Figure 2. Use of polypropylene suture to facilitate the anastomosis in cases of ruptured aneurysms or where difficult exposure is present.

ing of the graft if it is too long. Only a short tube portion is required if a bifurcation graft is used. Severe, constant pain and a cool extremity with paresthesias and numbness postoperatively should be explored immediately without arteriograms. Thrombosis occurring immediately postoperatively is most likely due to technical error. Late thrombosis should be studied with arteriograms, and infection should be suspected.

False aneurysm may occur from suture line disruption or infection. Suture line disruption may result from tension on the anastomosis, hypertension in the postoperative period, defect in the graft, friable aortic tissue, or with the use of silk suture in the anastomosis. Dacron pledgets are useful for reinforcement of the suture line in a friable aorta. Large bites on the aorta with a large (3-0 or 4-0) polyester suture minimizes tearing of the proximal anastomosis. To prevent subsequent dissection, all layers of the aorta need to be incorporated into the graft. A short cuff of graft material passed like a ring over the graft after the proximal anastomosis is completed can be used to support the suture line *(Figure 3)*. Finally, reinforcement of the proximal suture line with a free graft of omentum when needed, as well as covering the entire graft with aortic aneurysmal wall, will protect the duodenum and minimize disruption of the suture line and aortoduodenal fistula.

Infection presents after wound complications as hemorrhage, thrombosis of the graft, or false aneurysm. Prophylactic antibiotics are indicated, particularly if the graft passes to the groin. Performance of concomitant procedures, particularly where

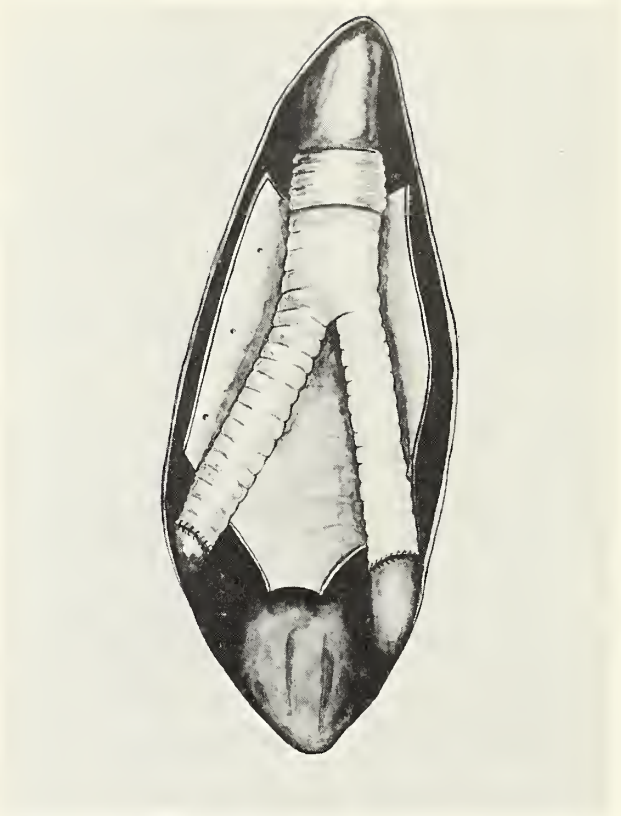


Figure 3. A cuff of graft material may be used to support the proximal suture line.

small or large intestine is opened, increases the risk of infection, and this should be avoided. If an unsuspected malignancy is found during laparotomy, the management depends on the underlying risk from each disease. Both should not be undertaken at the same time because of the high risk of infection.[12] Treatment of an infected graft requires removal of one limb or the entire graft with replacement through an uninfected area.

Ischemic colitis is seen infrequently, but problems are potentially avoidable. A large, tortuous inferior mesenteric artery on angiogram or at operation suggests that the superior mesenteric artery is occluded, and an attempt at reimplantation of the inferior mesenteric artery with a button of tissue should be considered. In special cases, preservation or reimplantation of one internal iliac artery may provide additional collateral circulation.

Prolonged ileus can be minimized by protecting and gentle handling of the small bowel when it is eviscerated. Retroperitoneal hematoma may also aggravate postoperative ileus. Distal embolization is prevented by minimal dissection initially, placement of distal clamps first, and appropriate flushing of the graft. Declamping shock is a result of hypovolemia

from hypoxic, dilated lower extremity arteries and release of lactic acid which has accumulated in the legs through anaerobic metabolism during cross-clamping. Volume replacement and bicarbonate administration is appropriate treatment.

Acute tubular necrosis is rarely seen today because of present fluid regimens.[13, 14] No incidence of renal failure requiring dialysis was seen in our elective, non-ruptured cases following this fluid regimen. Sexual dysfunction has been minimized using the intrasaccular approach.

Summary

The surgical management of AAA has been well outlined by others. Because of referral patterns in community hospitals, surgeons with variable vascular experience may not see a large number of cases. Based on the results of this series of AAA, guidelines for surgical management and technical points for avoidance of pitfalls are provided.

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Hyperparathyroidism

Clinical Manifestations of Parathyroid Adenomas

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PARATHYROID GLANDS were first described by Remak in 1855. Owen, in 1862, identified parathyroid glands in the rhinoceros and Virchow, in 1863, found them in man. In 1879 Sandstrom, in the dissection of a dog neck, observed structures he called "glandulae parathyreoidaea." The following year he performed fifty human dissections, finding the same glands. The relationship between parathyroid glands and calcium metabolism was recognized in 1906 by Erdheim. Parathyroid tumor as the cause of osteitis fibrosa cystica, rather than the result, was shown in 1915 by Schlagenhaufer. The first adenoma removed was in 1925 by Mandel. DeBois performed the first mediastinal parathyroid adenectomy in 1926 in New York.[1]

Embryologically the parathyroid glands develop from the third and fourth branchial pouches. Parathyroid three migrates below parathyroid four to become the inferior parathyroid. The embryologic association of parathyroid three with its branchial pouch counterpart thymus causes it to assume a variable position in the neck or superior mediastinum.

Nearly three-fourths of the superior parathyroid glands come to rest on the posterior surface of the middle third of the thyroid gland. More than one-half of the inferior parathyroids come to rest on the posterior surface of the lower pole of the thyroid gland or just below it.[2]

Calcium homeostasis is dependent upon parathormone from the parathyroid glands, vitamin D synthesized by the kidney, and calcitonin secreted by the "C" cells of the thyroid. Parathyroid hormone has two target organs — bone and kidney. It induces osteoclastic activity causing bone resorption and calcium liberation. This process is vitamin D dependent. The effect on the kidney is three-fold: it controls calcium excretion; it blocks tubular reabsorption of inorganic phosphate (increasing phosphate excretion); and it stimulates the synthesis of 1, 25 dihydroxy vitamin D-3. In addition, the kidney is the most important site of parathormone metabolism (degradation).

The active form of Vitamin D is 1, 25 dihydroxy vitamin D-3. In the presence of parathormone it stimulates calcium adsorption from the intestine and mobilization from the bone.

Adenoma of the parathyroid interferes with calcium homeostasis, resulting in a variety of manifestations of hyperthyroidism. Several cases are presented, and diagnosis and management are discussed.

Calcitonin acts on bone accretion, thereby significantly reducing the plasma concentration of calcium. This occurs by initially decreasing the number of osteoclasts, and later by increasing the number of osteoblasts.[3]

Hyperparathyroidism may be primary or secondary. Primary hyperparathyroidism is most often due to adenoma but may be caused by primary hyperplasia or carcinoma.

Secondary hyperparathyroidism results from prolonged or severe resistance of target organs to parathormone. This results in hypertrophy of the parathyroid glands and consequent excessive secretion of parathormone. Even if the resistance to the hormone is removed, it is by no means certain involution of the hypertrophied gland will occur.

It has been presumed in the past that some hyperplastic glands in secondary hyperparathyroidism develop into autonomous adenomas. This condition is called "tertiary hyperparathyroidism."[4]

Clinically, hyperparathyroidism has been said to lead to problems with "bones, stones, and abdominal groans." In addition, cardiovascular, neurologic, and ophthalmologic alterations may develop.[5]

During the past few years at St. Francis Hospital we have witnessed patients demonstrating each of these clinical manifestations; following are some illustrative cases.

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A 40-year-old white female was admitted complaining of the acute onset of right lower quadrant pain with associated nausea and vomiting. Physical examination disclosed right lower quadrant tenderness. The admission diagnosis was probable appendicitis. Examination of kidney, ureter, and bladder revealed no abnormalities except for some calcifications in the kidney areas. During the night the patient passed a renal stone, and an intravenous pyelogram the following day showed some dilatation of the ureters with the right ureter being dilated greater than the left. Radiographic evidence indicated that she had passed a kidney stone recently from the right ureter. Subsequent workup revealed calciums of 13.0, 13.6 and 14.7 mg/100 ml. A parathormone level also was obtained which was 190 μl Eq/ml with a normal of 0-40 μl Eq/ml. Surgical neck exploration revealed an 1100 mg chief cell adenoma of the parathyroid. The patient did well during the postoperative period with normal calciums and was discharged on the fifth postoperative day.

Originally, hyperparathyroidism was thought to be a disease of the bone. Osteitis fibrosa cystica is seldom seen today, and osseus manifestations are more subtle — such as resorption of the lamina dura of the teeth, subperiostial resorption of the phalanges, and bone cysts (''brown tumors'').

Hyperparathyroid patients sometimes present with abdominal pain. A 63-year-old white male was admitted through the emergency room complaining of severe epigastric pain without radiation, nausea, vomiting, and fever. Initial examination revealed a temperature of 38C, a markedly tender epigastrium, and hypoactive bowel sounds. The patient's hemoglobin on admission was 17.1 gm/100 ml, and he developed further hemoconcentration during his hospital course. His amylase likewise was elevated and continued to be high. In the face of acute pancreatitis and hypoalbuminemia, the patient still had a calcium of 12.0 mg/100 ml, which rose to 13.0 mg/100 ml on the third hospital day. On that day he became confused, hypotensive, and later suffered a respiratory arrest and died. At the time of autopsy the following pathologic findings were seen: examination of sections of pancreas showed inflammation, fat necrosis, and calcification with saponification. Examination of the neck disclosed a 400 mg tumor at the left lower border of the thyroid gland. Microscopic examination revealed a chief cell adenoma pushing normal parathyroid tissue peripherally as the adenoma increased in size.

Neurologic manifestations of hyperparathyroidism vary from mild to severe. A 44-year-old white male was admitted to the internal medicine service complaining of dizziness and light headedness. He had experienced three similar episodes during the six weeks prior to admission. He also complained of intermittent numbness of the left side of his face and right arm. His wife stated that for the previous six months he had been quite irritable. In the office his calcium was 12.3 mg/100 ml and two other calciums in the hospital were elevated. A blood sample was drawn and sent to the Mayo Clinic laboratory for evaluation of parathormone level. This revealed a PTH of 63 μl Eq/ml — in the range consistent with a primary hyperparathyroid condition. The patient was taken to surgery and a left inferior parathyroid adenoma was removed. It weighed 1560 mg. The patient did well during the postoperative period and was discharged on the fourth postoperative day with no medications.

Five to ten per cent of our patients had multiple system complaints. A 43-year-old white female was admitted to the hospital with hematemesis. Examination revealed epigastric tenderness and fullness. She also had a well healed upper abdominal surgical scar. On evaluation, her past medical history was remarkable; two years prior to admission she had undergone a subtotal gastrectomy for duodenal ulcer disease, and one year later she had been admitted to the hospital on the psychiatric service with a diagnosis of ''involutional depression.'' The psychiatrist noted that she had experienced a 20-30 pound weight loss, suffered from insomnia, anorexia, restlessness, morning anxiety, loss of interest in other people, and self-deprecatory feelings. Minnesota Multiphasic Personality Inventory revealed ''severe depressive reaction.'' The patient was treated at that time with thioridazine, trifluoperazine, chlorpromazine, and seven electroconvulsive therapies. During the current admission an upper gastrointestinal series revealed a previous gastrectomy and the suggestion of a marginal ulcer. Surgery disclosed a marginal jejunal ulcer with perforation into the left lobe of the liver and pancreas, and into multiple loops of jejenum and the transverse colon with a small gastrocolic fistula. Marked stenosis of the gastric outlet likewise was identified. Accordingly, a bilateral truncal vagotomy, gastrectomy, splenectomy, colon resection, and lysis of adhesions were performed. During the patient's postoperative convalescence it was noted that her serum calciums were elevated. During subsequent surgery a left inferior parathyroid adenoma weighing 1850 mg was removed. Her postoperative calciums returned to normal and she was discharged from the hospital in a much improved condition.

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Peritoneovenous Shunt

Early Functional Failure

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Peritoneovenous (PV) shunt placement in patients with medically intractable ascites results in beneficial effects for the patient such as improved nutrition and vigor, decreased abdominal girth, and improved diuresis. The procedure is illustrated in *Figures 1-7*. Laboratory follow-up showed increased tissue oxygenation, increased endogenous creatinine clearance and sodium excretion, increased plasma volume, cardiac output and glomerular filtration rate, decreased secondary hyperaldosteronism, renin and angiotensin secretion, reversal of hepatorenal syndrome, and decreased portal hypertension. Continued clinical and laboratory improvement was the best indication of shunt patency and its adequate function. Recurrent clinical and laboratory deterioration led to a presumptive diagnosis of shunt failure. Although variable results have been obtained by different observers, up to 57 per cent of PV shunts failed during serial observation, with up to 20 per cent of failures occurring almost immediately after shunt placement. Clinical findings alone were insufficient to distinguish anatomic failure (clotted shunt) from functional failure (abolished PV pressure gradient). Interest in distinguishing between the two types of failure is more than academic, because each failure requires a specific therapeutic approach. Correction of anatomic shunt failure requires reoperation for partial or total replacement of the obstructed shunt. Correction of functional failure could be accomplished by non-invasively re-establishing PV gradient.

In a prior communication we emphasized the use of a Doppler ultrasound flowmeter to differentiate late functional and anatomic failure. The present study was conducted to evaluate the usefulness of this instrument in detection of early (intraoperative) shunt failure.

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Case Report

A 72-year-old obese female, with past history of chronic alcoholism and biopsy-proven Laennec cirrhosis, presented with anasarca, intractable ascites, oliguria, deteriorating renal function, weakness, and severe respiratory distress. Vital signs were unstable. Intraperitoneal pressure was 18.7 cm H_2O; central venous pressure was 5 cm H_2O. After an unsuccessful trial of energetic medical treatment, a PV shunt (LeVee shunt) was placed using local anesthesia. The multifenestrated abdominal limb was placed intraperitoneally in the right upper quadrant between the liver and the colon. The pressure-sensitive one-way valve was placed subperitoneally and the venous limb was advanced subcutaneously to the neck and introduced into the internal jugular vein. Fluoroscopy showed the tip of the venous limb to be in correct position. A sterile 9.3 MH_z pencil Doppler probe, connected to a model 810 Parks Electronics Flowmeter, was used to detect ascitic flow through the subcutaneous tubing, as soon as the shunt had been placed. Ascitis flow was easily detected. Closure of the abdominal and neck wounds was carefully completed to minimize ascitic leakage and — in the case of the abdominal wound — to avoid incisional herniation. Within ten minutes of shunt placement, ascitic flow could no longer be detected. Central venous pressure was again measured and was 18 cm H_2O. Intraperitoneal pressure was 9 cm H_2O. The cervical wound was partially reopened and the shunt was transiently clamped with a vascular clamp (Softjaws, Edwards Laboratory), while intravenous cardiotonics and a furosemide diuretic (Lasix) were promptly administered. Within 30 minutes, there was a diuresis of 600 ml and the

Use of a peritoneovenous shunt provides significant benefits for patients with medically intractable ascites. The Doppler ultrasound flowmeter — previously determined to be useful in differentiation between late functional and anatomic shunt failure — is here evaluated for usefulness in the detection of early (intraoperative) shunt failure.



Figure 1. Indication for PV shunting: intractable ascites.



Figure 2. The peritoneal limb and the valve of the shunt have been placed. The subcutaneous tunnel for the venous limb is being made.



Figure 3. The venous limb has been placed in the subcutaneous tunnel.



Figure 4. The venous limb is seen riding upon the clavicle and reaching into the internal jugular vein.



Figure 5. Abdominal incision closure.



Figure 6. Neck incision closure.



Figure 7

central venous pressure had decreased to 12 cm H_2O. The shunt was unclamped, and the cervical wound was closed. Ascitic flow could again be detected in the subcutaneous tubing by Doppler ultrasound flowmetry. Central venous pressure continued to oscillate between 10 and 14 cm H_2O within the first five hours following operation, then dropped to 3-5 cm H_2O. Increased diuresis persisted, whereas respiratory distress and abdominal girth decreased remarkably. At the end of the six-day observation period, there was continuing clinical improvement and persisting ascitic flow through the shunt, as detected by Doppler ultrasound flowmetry. Three months after shunt placement the patient was ambulatory, stronger, had decreased abdominal girth, good renal function, and a 30-lb weight loss. Doppler testing showed continuing shunt function.

Discussion

Pronounced PV gradient produces a sudden and massive ascitic flow into the venous system, thus leading to central venous hypertension and intraoperative functional shunt failure. Partial external drainage of ascites before opening of the shunt could prevent this sequence of events, thus minimizing the risks of subsequent congestive heart failure and acute pulmonary edema.

In absence of these complications, several hours after successful shunt function, PV pressure gradient decreases and leads to decreased ascitic flow through the shunt. Later, flow becomes intermittent, and usually after 10 days no ascitic flow can be detected by Doppler ultrasound flowmetry, in absence of any ancillary PV gradient increasing maneuvers. To promote continuing shunt function, long-term use of an abdominal binder and respiratory exercises (inspiration against resistance) are recommended. To detect ascitic flow in the shunt by Doppler flowmetry, transient manual abdominal compression is advised.

In absence of any positive Doppler flow signal, further diagnostic investigation is necessary to differentiate functional from anatomic shunt failure.

Radionuclide detection of shunt patency is the next diagnostic step. Tc-99m sulfur colloid or Tc-99m albumin can be utilized for this test in doses ranging from 2-10 m Ci according to various investigators. The isotope is injected into the peritoneal cavity and the patient asked to perform breathing exercises. Serial abdominal and anterior chest wall scintiscans are performed with an Anger Camera to monitor isotope mobilization. According to early observers (Kirchmer *et al.*[1] and Gorten[2]), isotope concentration by the liver or spleen and its clearance from the peritoneal cavity within the first two hours of intraperitoneal isotope injection is an indication of adequate shunt function. The validity of such observations, which were based upon single case reports, has been questioned by Pujara and Russell.[3] Isotopic visualization of the subcutaneous catheter and of the heart and large vessels is a better indication of shunt function.[4] If no isotopic visualization of these structures occurs, functional or anatomic shunt failure can not be differentiated from each other with this method.[5]

Radiological detection of shunt failure is a more definitive but more invasive method because it requires injection of hydrosoluble contrast medium in the venous limb of the shunt.[6] Ten ml of 50 per cent renographin is injected by puncturing the silicone tubing below the clavicle with a gauge #25 needle, and films are taken immediately and at regular intervals up to 20 minutes later.[5] This method can show persistent shunt patency, shunt obstruction, or shunt malposition. Because puncture of the shunt is required, serial studies carry a risk of shunt tubing deterioration and backflow of blood into the shunt leading to shunt obstruction. There is increasing evidence that PV shunting produces disseminated intravascular coagulation.[7-10] Falling platelet counts and rising PT, APTT, and fibrin degradation products are indicative of continuing shunt patency[11] whereas parameter normalization follows shunt obstruction.[7] If further experience corroborates these findings, such results could be used as a criteria to determine shunt patency.

Critical evaluation of methods for detection of shunt patency shows that contrast radiography is the

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Sunscreens

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THE SUN is not the unmixed blessing travel agents and sunbelt denizens would have us believe. Aside from evoking distinctive cutaneous reaction patterns in disease states (lupus erythematosus, porphyria, pellagra, and polymorphous light eruption) and in chemically induced photosensitivity states which are secondary to internal or topical application of chemicals, it plays an important role in three conditions almost all of us face in one degree or another: sunburn, aging, and skin cancer.

Sunburn

In all but the most severe forms, sunburn simply means skin erythema from sunlight — erythema solare. There appear to be two components to this reaction: (1) an early evanescent erythema that disappears a few minutes after exposure; and (2) a more persistent, delayed erythema that peaks 12-24 hrs later and then fades in two to three days. Vasodilatation and leukocyte chemotaxis, together with variable degrees of edema formation, underlie the clinical changes.

The skin contains many light absorbers — chromophores — such as melanin, nucleic acids, amino acids, and tetrapyrroles. After light is absorbed, complex photochemical reactions take place which probably release multiple inflammatory mediators, including histamine, kinins, and prostaglandins. Much more needs to be learned about the molecular mechanisms involved in sunburn.

Photobiology

Three important photobiological terms should be defined when discussing sunscreens: ultraviolet B (UVB); ultraviolet A (UVA); and minimal erythema dose (MED).

UVB is that portion of the sun's electromagnetic spectrum with wave lengths ranging from 290-320 nanometers (nm). These rays are responsible for most of the photobiological reactions that occur in ordinary, untreated skin: sunburn, suntan, vitamin D synthesis, and — most probably — aging and skin cancer.

UVA includes wave lengths from 320-400 nm, where visible light begins. The effects of UVA on skin are less well understood than those of UVB, but they probably have an enhancing or augmenting effect on UVB-induced skin damage. For all practical purposes, solar wave lengths shorter than 290 nm do not reach the earth's surface.

MED refers to the lowest dose of UVB — generally measured in seconds or minutes depending on the light source — that produces a barely perceptible erythema in normal untanned skin 24 hrs after exposure. For Caucasians, this is usually 15-30 minutes of midday sun.

T. B. Fitzpatrick of Harvard suggests that the MED for a given individual may be predicted according to his or her anthropologic origins. Those with the shortest MEDs, and thus the lowest thresholds to sunburn, are designated Skin Type I, and those with the longest MEDs, the highest thresholds, are designated Skin Type VI, with four intermediate types designated II-V.

Sunscreens

Three main factors require consideration when selecting or evaluating a sunscreen: (1) absorptivity

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— how effectively the agent screens out ultraviolet radiation; (2) substantivity — how well the material adheres to the skin despite sweating and bathing; and (3) cosmetic appeal. Certain so-called physical sunscreens or sunshades (titanium dioxide, zinc oxide) are quite effective at blocking the passage of light, both visible and ultraviolet, but simply are not practical for widespread, everyday usage, except over small areas such as the nose, because they are opaque.

The Food and Drug Administration (FDA) has recently adopted a numerical system of designating the degree to which various commercial sunscreens absorb UVB — a kind of efficiency rating. Package labels now display the sunburn protection factor (SPF) of each product. The SPF actually represents the ratio between the MED for sunscreen-treated skin and the MED for untreated skin. Thus, the higher the SPF value, the more efficient the sunscreen is at raising the threshold to UVB-induced erythema. The highest SPF value the FDA currently allows is 15. An SPF of two implies that as much as 50 per cent of incident UVB is transmitted through the sunscreen *(Table I)*.

Substantivity data are also available for most sunscreens but have not been as widely promoted as the SPF system in the marketing and advertising of these products. Regardless of the specific product and its substantivity claims, it is generally advisable to wait one or two hours after application before going out in the sun, showering, swimming, or exercising; but effecting compliance with this recommendation is often difficult, especially with children.

Chemical sunscreens — those that absorb and do not simply physically block the passage of light — fall into one of three broad categories: (1) para-aminobenzoic acid (PABA) and its esters; (2) other substituted non-PABA benzoates such as cinnamates, homosalate, and anthranilate; and (3) benzophenones. In general, PABA and its esters per-

TABLE I
COMMERCIALLY AVAILABLE SUNSCREENS AND THEIR RELATIVE PROTECTION, SUBSTANTIVITY, AND COST RATINGS

Product (Manufacturer)	*Active Agent(s)*	*SPF*	*Substantivity*	*Relative Cost**
Piz Buin Exclusiv Extrem†	cinnamate, benzophenone, imidazole	15	excellent	?
Coppertone Super Shade 15 (Schering-Plough)	padimate O, benzophenone	15	excellent	moderate
Total Eclipse (Herbert)	glyceryl PABA, padimate O, benzophenone	15	excellent	high
PreSun 15 (Westwood)	PABA, padimate O, benzophenone	15	good	moderate
Pabanol (Elder)	PABA	14	good	moderate
Sunbrella (Dorsey)	PABA	10	good	high
Original Eclipse (Herbert)	glyceryl PABA, padimate O	10	fair	moderate
Block Out (Smith Kline)	padimate O	10	?	high
RVPaque (Elder)	red veterinary petrolatum, cinnamate, zinc oxide	10	poor	high
A-Fil (Parke-Davis)	anthranilate, titanium dioxide	8	fair	high
Maxafil (Parke-Davis)	anthranilate, cinnamate	8	fair	high
Piz Buin 6 (Westwood)	cinnamate, benzophenone	6	good	moderate
Sundown Extra Protection (Johnson & Johnson)	padimate O, benzophenone	6	good	moderate
pabaGel (Owen)	PABA	6	fair	moderate
Sea & Ski (Smith Kline)	padimate O	6	poor	low
Sun Gard (Dome)	padimate O, benzophenone	6	poor	moderate
Piz Buin 4 (Westwood)	cinnamate	4	fair	moderate
sunDare (Parke-Davis)	cinnamate	4	?	low
UVAL (Dorsey)	benzophenone	4	poor	high
Coppertone 2 (Schering-Plough)	homosalate	2	poor	low

* These cost estimates, although derived in large part from published indices (See *Facts and Comparisons)*, are admittedly somewhat arbitrary. "High" generally means $1 or more per ounce retail price, "low" 50¢ or less per ounce.

† This highly promising sunscreen has been available for several years in Europe but, unfortunately, is not yet on the American market.

form best against UVB; their absorption spectrum is relatively narrow but highly efficient. The non-PABA sunscreens offer less protection than PABA against UVB but absorb over a broader range into the UVA range. Protection against UVA, which is not reflected in a sunscreen's SPF rating, is desirable for patients taking potentially photosensitizing drugs.

Some newer products combine different chemical classes of sunscreens and thus seek the best of both worlds: high absorptivity and a broad absorption spectrum. One such combination called for the sequential application of two separate agents — dihydroxyacetone and hydroxynaphthoquinone (Duoshield) — and held some promise in the treatment of protoporphyria patients, but it was discontinued by the manufacturer. Another combines an imidazole derivative with a cinnamate and a benzophenone (Piz Buin Exclusiv Extrem Creme) and claims excellent substantivity, high SPF (15), and breadth of absorption (UVA as well as UVB).

Used alone, non-PABA benzoates and benzophenones tend to have less substantivity than PABA and its esters. Stinging sensations are a frequent complaint with benzophenones, PABA esters, and hydroalcoholic PABA, especially if the skin is already inflamed. However, benzophenones and non-PABA benzoates cause less staining of clothing than PABA.

Early formulations containing PABA esters did not seem to perform as well as the parent PABA in alcohol formulation, but with improvements in the base, they now seem to match or even exceed SPF and substantivity of hydroalcoholic PABA. The two most popular esters are amyl dimethyl PABA (Escalol 506, padimate A) and octyl dimethyl PABA (Escalol 507, padimate 0). Padimate O may be safer than padimate A in terms of not causing a photodermatitis; some authorities believe that padimate A is inherently photosensitizing. Glyceryl PABA (Escalol 106) has received some negative publicity as a contact allergen and photo-allergen, but evidence for this is inconclusive because some of the older preparations of glyceryl PABA were contaminated with benzocaine, a notorious contact allergen.

Table 1 lists the more commonly used topical sunscreens available without prescription. Combination products with an SPF of 10 or greater appear to be in the forefront, but PABA and padimate O also absorb UVB very well as single agents. Unfortunately, higher SPF and substantivity ratings also tend to be associated with higher retail prices.

A sunscreen with an SPF of 8 or higher is probably best for individuals with skin types I, II, and III (easy burners) who are choosing a sunscreen for the first time. Good sunscreens found in the United States include Total Eclipse, Super Shade 15, and PreSun 15. For those who sunburn less readily — types IV and V — sunscreens with SPFs of 2-6 probably provide adequate protection. For blacks and aborigines — types VI — sunscreens are not ordinarily indicated. Caucasian children generally require SPFs of 6 or greater.

Adverse Reactions

Stinging of skin, contact sensitization, and contact photosensitization have been mentioned as possible adverse reactions to sunscreens; these are especially apt to occur with PABA-type compounds, notably glyceryl PABA. Before recommending PABA or its esters, inquiry should be made as to history of allergy to hair dye (para-phenylenediamine) or to PABA-type anesthetics (procaine, benzocaine) because cross-reactions may occur. If irritation or stinging is unavoidable, as in some photosensitive eczemas, combining a sunscreen with a topical corticosteroid is often useful. It is interesting that physical sunscreens do not appear to be sensitizing.

Until recently, another form of photosensitization which sometimes occurs received little attention: photoallergy due to fragrances. 6-Methylcoumarin has been liberally added to a number of less ethically promoted products, mostly suntan lotions, to impart a lush, fruity scent. This agent is now known to be a fairly strong contact photosensitizer. This possibility should be considered in any individual who develops an eczematous dermatitis in exposed areas following usage of one of these cosmetics. Wherever possible, formal phototesting can be used to confirm the diagnosis.

Systemic Photoprotectants

For the most part, attempts to formulate a safe and effective internal photoprotectant have been disappointing. Many agents have been tested, including PABA, aspirin, and vitamins A, C and E, but few have lived up to the claims. Oral beta-carotene (Solatene), however, has given substantial protection from photosensitivity in erythropoietic protoporphyria. Some cases of polymorphous light eruption also have been reported to benefit from beta-carotene. On the whole, its usefulness remains limited.

Summary

The search for a completely safe, effective, and affordable topical sunscreen continues. In the meantime, physicians should encourage regular use of an appropriate sunscreen for patients whose skin tends

to be harmed by repeated, excessive sun exposure.

Self-Assessment Questions

1. All of the following are induced most efficiently by UVB as opposed to other wave lengths. The exception is:
 a. sunburn
 b. suntan
 c. skin cancer
 d. vitamin D synthesis
 e. protoporphyria skin lesions
2. All of the following are properties of UVA. The exception is:
 a. transmitted through window glass
 b. responsible for weak or transient tanning
 c. responsible for most cases of chemical photosensitization
 d. visible to the naked eye
 e. enhances skin-damaging effects of UVB
3. All of the following products contain only PABA or padimate and therefore absorb principally UVB and comparatively little UVA. The exception is:
 a. Block Out
 b. Total Eclipse
 c. Sunbrella
 d. pabaGel
 e. Pabanol
4. Which one of the following products appears to have the highest substantivity?
 a. Sea & Ski
 b. Coppertone 2
 c. Coppertone Super Shade 15
 d. SunGard
 e. UVAL
5. Which one of the following products is most likely to produce an allergic contact dermatitis in an individual sensitive to benzocaine?
 a. Sea & Ski
 b. Coppertone 2
 c. Coppertone Super Shade 15
 d. Total Eclipse
 e. Block Out

(Answers on page 259)

Peritoneovenous Shunt

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most accurate one but also prone to complications not occurring with the other methods. Because of its simplicity, we feel that Doppler ultrasound flowmetry is the best one for initial screening purposes.

This test does not require extensive displacement of instruments or patients, it can be performed immediately by the physician in charge of the patient, and it can be repeated as many times as necessary without risk or pain to the patient or invasion of the shunt. If ascitic flow is detected, no further studies are necessary. If ascitic flow is not detectable, radionuclide or radiographic techniques can be used.

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Medical HISTORY

The Philosophy of a Neurosurgeon

LOYAL DAVIS, M.D.,* *Phoenix, Arizona*

Neurological surgery became a special field of surgery in the United States when Harvey Cushing convinced his doubting professor of surgery at Johns Hopkins Hospital — William Stewart Halsted — that he could successfully devote himself to the surgical treatment of diseases of the nervous system. In Great Britain, Henry Head and Hughlings Jackson, neurologists; Charles Sherrington, neurophysiologist; and Victor Horsley, surgeon, had aroused and stimulated Cushing's interest in a field of surgery to which no one in the United States had been attracted.

Neurological surgery in the United States was further advanced by treatment of wounds during World War I. The two outstanding contributions in neurological surgery from that war were Harvey Cushing's treatment of head injuries and Lewis Pollock's study of peripheral nerve injuries. Then a group of surgeons, some of whom were veterans of military service during World War I — Charles Elsberg, New York; Charles Frazier, Philadelphia; Allen Kanavel, Chicago; and Howard Naffziger, San Francisco — became interested in the surgical treatment of diseases of the nervous system. All had been educated and trained as general surgeons. They applied the principles of surgery successfully in their operations. They depended greatly upon the diagnostic talents of their associates in neurology. In the beginning, the diagnoses were based entirely on clinical acumen in evaluating symptoms and signs. The exquisite abilities of neurologists like Hugh Patrick, Chicago; William Spiller, Philadelphia; Frederick Tilney, New York; Edward Taylor, Boston; and Lewis Pollock, Chicago, have been replaced by more and more mechanical diagnostic methods as these practitioners of the art of neurology disappeared.

In the 1920s, there were no surgical residency educational and training programs in the Midwest; there were only a few dedicated surgeons who voluntarily assumed the responsibility for teaching and training young men in the principles and techniques of surgery. Such a man was Allen Kanavel, who financed and guided several young men through a progressive four-year program of surgical education. There were other surgeons who utilized the services of young men in their practice without the primary purpose of educating them. They often stipulated that when either party to the association decided there had been a sufficient osmotic transference, the young man had become a competitor and could not practice within a specified geographic radius of their office. It was this type of assistantship — consisting primarily of service rather than education — that unfortunately brought the term "preceptorship" into disrepute. The definition of "preceptor" is "teacher," and this means the acceptance of responsibility for the preceptee or pupil — *i.e.*, a personal interest in the product of the teacher's efforts. Such personal interest requires energy, talent, ability, intellectual endeavor and sacrifice from the teacher, and a reciprocal expenditure by the pupil. The teacher's interest in his pupil's professional and personal life should never cease to be helpful.

Neurosurgical residency programs should be based on a progressive, planned course of instruction and training. It is postgraduate education at the highest level because human life is involved. With unprecedented progress in all phases of medicine, training and excellence in the performance of surgical procedures alone are no longer sufficient. A curriculum that is broad, progressive, and includes the basic fundamentals of surgery must be completed as a first step by the candidate. The faculty of a medical school should delineate in detail its curricular requirements in neurosurgical education, as in other areas of graduate study. In bridging the gap between the sciences and the humanities, the faculty in phi-

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losophy, mathematics, languages, and history can participate and contribute in the broadest sense in the education of a neurological surgeon, but by necessity, the program must be focused on the hospital and the laboratories of the medical school.

The surgical faculty of the medical school should state that the candidate has fulfilled the prescribed course of graduate study in a satisfactory manner, that he has been examined by them and has been granted an advanced degree, indicating that they believe him to be a thoroughly and properly educated surgeon. Further, the faculty should state that the candidate is prepared for an examination of his qualifications as a neurological surgeon. Perhaps one day some imaginative young professor of neurological surgery will announce such a program, disregarding the strictures of the examining board of neurological surgery, and prepare his graduate students to challenge the examiners.

In 1912, it was suggested by the founder of the American College of Surgeons (ACS), Franklin H. Martin, as one of the platforms for improving the care of surgical patients, that the faculties of medical schools assume the responsibility for such graduate education. This responsibility has not generally been accepted by university faculties in the United States; they appear content to delegate the education and training of surgeons to individual hospital surgical staffs. Gradually, medical school staffs at universities have relinquished what little influence they have had in the determination of the character and maintenance of the quality of surgical education to groups that should function as bodies of completely independent and unprejudiced examiners. These examining bodies should not be responsible for the review, approval, or disapproval of the content and conduct of graduate study in surgery. In recognition of completion of the course of graduate neurosurgical study, and successful passing of the board examination, Fellowship in the American College of Surgeons should be granted after a satisfactory demonstration of, adherence to, and support of the principles of ethical and professional practices and conduct promulgated by that College, and in recent years this has transpired.

The Committee on Graduate Training in Surgery, the Council on Medical Education, the Advisory Board for Medical Specialties, and the Boards of Surgery and Surgical Specialties unquestionably have had an important influence on the improved standards of education for surgeons, thus elevating the care of the surgical patient. It is time, however, for universities and their medical school faculties to play a more important role — a very definite and dominant role — in the education of surgeons. Too often, neurosurgical residency programs are loosely referred to as "training" whereas such programs should be a comprehensive period of graduate education in the surgical care of sick patients in which operative techniques are an integral but not the totally dominant content.

Neurological surgery should be taught by surgeons dedicated to the teaching of young physicians; the type of personal teaching that has been given and received successfully in the past included the patient who voluntarily selected the surgeon and paid a fee for such services. In fact, in many instances, the young surgeon's education came exclusively from such patients. Those surgeons invariably instructed their resident students personally, and delegated increasing responsibility so that the patient was well aware of the high quality of care he/she received, and shared with pride in the progressive type of education of the younger physician. No premium should be placed upon the number of operations the resident performs independently. He/she should be taught consistently, day by day, operation by operation, the correct and accepted surgical methods, as well as pre- and post-operative procedures. When the resident has the opportunity to act as the operating surgeon, his surgical teacher should serve as his first assistant. The resident should not be permitted to learn by operating without direct supervision, because the goal of his teacher should be to insure the patient the best possible care, and at the same time teach the student.

Increasingly, those responsible for administering departments of neurological surgery appear to believe that their surgical reputation depends upon their peripatetic wanderings and a talent for obtaining government grants. The products of their teaching programs would be improved if they devoted themselves to encouraging clinical investigation, carrying on their own experimental research, and treating patients. They speak authoritatively about spinal cord injuries, for example, when the number of civilian patients they have encountered is few. These are academic neurological surgeons, a term designed to elevate their status above that of the surgeon who treats patients. The resident is given more and more latitude, often when he is not capable. Numbers of operations have become more of a criterion of accomplishment than well-performed operations carefully supervised. It is easier for residency review committees to play the numbers game than to judge the quality of the resident's education.

It has been incorrectly assumed, and definitely and defensively claimed, that close supervision in-

terferes with the resident's progress in acquiring independent responsibility. This philosophy tends to assign the patient a secondary role instead of fulfilling the primary objective of providing the patient with the best surgical care. The patient is, and should be, the primary concern of the surgeon; he is not the jurisdictional right of members of the house staff who receive increasing remuneration for pursuing their post-graduate medical education and training; this is a complete reversal of their own responsibility for financing their medical school education, and is reflected in the patient's skyrocketing daily bed cost.

At present, there is a complete lack of respect for the attending surgeon by many young people who come into a hospital as students and members of the house staff. Their slovenly appearance — with dirty, long hair, offensive body odors, dirty hands and nails, and non-descript and inappropriate clothing — belies their professional medical goals, regardless of their intelligence. It is fashionable for them to speak of treating the patient as a whole — that is, to treat him psychologically — yet they lack the basic element to make the patient comfortable — that is, to look and act like a physician. These are the new young doctors who speak of an "interesting case" which allies them *with* a disease as well as *against* a patient.

A recent study of the American Surgical Association and the American College of Surgeons has revealed that there are 163 physicians/100,000 people in the United States. There are 95,000 licensed physicians who practice surgery; of these, 53,000 are qualified by education and training, and are board certified. Licensure by a state board of medical examiners gives every physician the right to operate upon anyone who is willing to be placed on the operating table. Medical licensure in the United States should be specific and restrictive as far as the practice of surgery is concerned. This study suggests that there are too many surgical and surgical specialty residency programs. This infers also that the selection of physicians to enter these programs was based on numbers of residents, conferring prestige, rather than upon carefully chosen selective criteria. The conclusion is inescapable that there are more neurological surgeons than the demands for care justify.

The reputation of a surgeon is more sensitive than that of any member of any other profession. His good name is exquisitely reactive to the gossip of patients and their relatives. It is quite simple for a surgeon to become careless and assume that patients know as much about their illness as he does; or to become frustrated after explaining carefully to them, in as simple language as he can use, to have them give evidence by their next question that they have comprehended little. It is easy to form the habit of telling them nothing and to rationalize that this is best for their psychological state. Indications for an operation, what is hoped can be accomplished, and the extent of the continued follow-up care should be explained in detail to the patient and his family. This should become a matter of record in a specifically dictated consent for operation. The most fantastic errors can be encountered in the verbal relay of this information. This, together with the lack of a carefully recorded examination signed by the surgeon, a personally dictated operation note, and an accurate follow-up record can form the basis for a successful malpractice suit.

There should be a discussion of the surgical fee — before or after the operation — taking into consideration the responsibilities of the family, the earning capacities, and their ambitions and aims. The patient should not be suprised by the amount of the bill received in the mail. The benefits of medical care insurance should be paid directly to the patient, and the fee for professional services paid to the surgeon by the patient. This strengthens an important part of the patient/surgeon relationship, and gives the patient the benefits to which he is entitled by payment of his premiums. Fee schedules have been set by insurance companies for various types of operations, and many are ridiculous. These fees are understood to be acceptable to the profession, regardless of the skill and talent required, or the economic ability of the patient to pay. It makes a trade union of a profession, and encourages the establishment of a level of mediocrity.

There is, of course, a difference in the patients of today and yesterday. It is too easy to place all the onus for a poor physician/patient relationship upon changes in the physician. With some modern patients, a major surgical operation is followed by a minor degree of gratitude; the patient's life may be saved, only to have him complain that his sutures stick, the nurses have cold hands, the hospital bed is hard, the coffee is abominable, and the hospital bill and the surgeon's fee are outrageous. Today the patient may rush off to the law courts if he wakes up from an extensive life-saving operation and a safety-pin is sticking him, or if he develops a backache at work. The citizen has become more and more security-minded and more dependent upon his union and governmental agencies. The world, he says, owes him a living. What is, then, more natural than for him to turn to his surgeon's insurance company.

which appears to be a limitless source of wealth? He rationalizes by saying it won't take anything out of the surgeon's pocket.

The increasing interest of the public in medicine and a knowledge of medical facts are factors that exert a profound influence on the practice of surgery. Articles fill magazines and newspapers, and scenes of the operating room are as familiar as frontier saloons on television screens. The general effect of public interest and knowledge of medicine has been good, but often the popular presentation of a surgical procedure has been exaggerated to make it effective to the layman. The result may be that the reader or the viewer, who is not too discerning at best, may be misled to anticipate a more favorable outcome of an illness or injury than even the best surgeon or the best operation can deliver. Weekly picture magazines vividly portray the surgical anatomy, the pathology, and the operative steps in treatment of diseases so clearly that the patient may regard his surgeon as mentally retarded if he expresses a reservation.

There is no justification for the surgeon who releases an announcement of the results of experimental or clinical investigation, or an impending new operation to the press prior to presentation before a scientific society of his peers. He is helpless to stop the press from reporting upon his presentation at the society, but with the cooperation of his colleagues in the society, he can attempt to influence a reasonably factual report. However, regardless of all efforts, the headlines covering the story are neither under the control of the writer, the surgeon, nor the society. They are written for the single purpose of selling newspapers, and can therefore be entirely misleading to patients.

The United States is experiencing the trials, tribulations, and penalties of the era of the half-done job. To put it another way, this is an era in which there are constant attempts to legislate all facets of enterprise to the level of mediocrity. It is the age of plumbers who may come to fix a leaky faucet, someday — maybe; of salespersons who at their pleasure respond to inquiries about merchandise; of teachers who demand a single salary schedule so that achievement cannot be rewarded; of students who take cinch courses; and of physicians who sponsor fee schedules that reward the unqualified surgeon equally with the educated and trained surgeon. There appears to be a stampede away from the goal of first-rateness and responsibility. No one can take issue with the basic tenets of democracy that all men are born free and equal and are entitled to life, liberty and the pursuit of happiness, but it is manifestly absurd to maintain that a physician with no education and training in surgery, who calls himself a surgeon and who has the legal right to operate on a patient without restriction, is the equal of the surgeon who has voluntarily spent many years in formal education and training to become qualified to express an authoritative opinion which he can implement with skill and talent. Self-declared surgeons have attempted to establish certifying boards by edict with standards below the accepted minimum. Failing to receive acceptance, they used the goon tactics of labor union leaders to attempt to force their will upon those who strive to maintain first rate care for surgical patients.

To strive for the first rate in surgery entails the willingness to listen to the suggestions of the young, to modify and adopt them, if possible, in education, training, and practice. It does not mean, however, abandonment of experience and tradition, or substitution of the mediocre for the excellent. The pursuit of the first rate often encounters a philosophy that what is right is not as important as what is momentarily beneficial to the individual or the organization. There is reason to have knowledge of and respect for orthodoxy in the education, training, and practice of surgeons. This means holding the correct views that are in the best interest of the public. The care of the surgical patient is a matter of public interest; the difficulty is preventing orthodoxy from passing into stagnation or assuming prerogatives that do not permit discarding theories and practices that have ceased to be commonly accepted or have no further value.

Skill is in demand. How is it to be rewarded? Surgeons are skilled, and teachers are skilled. Both provide an essential service to the public; yet each profession has withdrawn that service at times for various reasons. Had the provocation been there 10 or 15 years ago, would they have rejected such action as contrary to their code of ethics? That they do so now is a measure of the frustrations of good men and good women faced with an economic climate of inflation, a dose of political dogma that largely denies merit its adequate reward, and engages in reckless deficit spending. The moment any member of the surgical profession gives way to the temptation to equate service with the hourly pay rate, the character of our democratic society is likely to be undermined beyond repair. If the surgeon and the teacher withdraw, what about others? This may lead to social anarchy and government control of all facets of life.

The President's Message

Medical marriages are said often to be in trouble because physicians who are autocratic, exacting and intense in the office, after working under demanding schedules with awesome responsibilities, fail to recognize that a different role must be assumed at home where there is a spouse who needs self esteem and there are children who need love, attention, and time. We all know how difficult it is to assume a different role when we leave our office or the hospital and find ourselves at home, at church, at a social gathering, or in a meeting.

When we gather as the Kansas Medical Society — whether in committee meetings, at the House of Delegates, or in testimony before the legislature — we may find it equally difficult to alter our approach to problem-solving and to leave the parochial interests of our specialties, our individual patients, our geographical areas within the state, or our personal financial security for the responsibility of helping to see that all Kansans have access to optimal health care.

If we are to be effective in our mission, we must recognize that organized medicine is more than the special interests of its individual physicians. When acting in concert we must develop a dominant broad point of view even though it will be finely tuned by our accumulated expertise as physicians who in another place and at another time give direct person-to-person care.



I look forward to working with you this coming year, and hope that together we can all rise above ourselves to meet our responsibilities to the Kansas Medical Society and the people of Kansas.

Fraternally,

President



Ads libitum

The two have had a long but somewhat erratic relationship, the one willing and able but with numerous other activities of greater promise, the other aloof and, save for occasional localized lapses, harboring a fundamental distaste for the former such as might be felt by a white-tied concert master for a garish, sequined rock star. But the advertising industry is enjoying an ascendancy such as it has never known before, and the medical profession is contending with new values imposed on time-honored principles (a condition it blames, with some justice, on the other), and the twain are meeting with disconcerting frequency.

True, physicians have never been far from those advertising techniques they found, if not fully compatible, at least tolerable — the occasional flamboyant personality attracting public interest and comment, the appropriately placed press notice of legitimate professional service, the name-paging (in pre-electronic days) at public gatherings, the subtle encouragement of word-of-mouth testimonials — all mechanisms by which name and reputation could be broadcast without incurring too much peer displeasure. Though such maneuvers are age-old, we suspect the restrictive policy of recent decades was seeded a century ago when the medical scene was congested with a bewildering variety of agents — allopaths, osteopaths, homeopaths, eclectics, chiropractors, naturopaths, herbalists, and so on. Imbued with a sense of greater qualification and higher calling, the orthodox physicians reacted against the excessive claims and promotional techniques widely used and became increasingly obdurate in their resistance to such activities.

The result has been a policy of almost complete restriction of publicity which has rested comfortably under the ethical cloak and at the same time simplified control since the totality of the restrictions eliminated any need to ponder the gray areas. Perhaps, too, it has been related to the increasing organization of the profession which has progressed from the near chaotic level of a hundred years ago to a complicated system of general and special groups united in disavowing overt advertising. Perhaps, in those days, everyone was too busy to feel the need to advertise or too preoccupied with issues considered of greater importance to give more than passing attention to the occasional transgressors. This is not to say the prohibition has been uncontested and accepted without dissonance or that the conforming physicians enjoyed a purity of character now being discarded by advocates of advertising within the profession.

Still, few features of medical behavior have persisted so long without concerted challenge — and the action, when it came, has been largely from the outside. It seems the social convulsions are throwing a bilious light on a whole catalog of earlier values. It is worth considering that the Federal Trade Commission, so frequently assigned the archvillain's role in the current medical morality play, has been, in fact, an agent of the forces that have been applying the new rules to the old players. A myriad of shifts in public and private thinking are changing attitudes toward the practice of medicine. For example, group practice at one time met with violent objection within the profession. One visible feature of the system was the impersonal label of a given group which, as the idea attracted public attention, permitted public reference to its activities much to the annoyance of individual practitioners. But the system has grown on its own merits and with it publicity techniques which are generally accepted and which, in turn, have conditioned the profession to the acceptance of a more liberal publicity policy.

We suggest, as well, the probability (though we make no claim for factual support) that the younger members of the profession are more interested in relaxing the bonds on advertising than are the older

ones. This is not intended to inspire any generational conflicts but to recognize their view of the world as compared with that of the older members (all right — *ours*). Not only do they see the obvious effect of practice promotion (free from the stigma of earlier days), but the rigidity of the establishment makes it, as always, an inviting adversary. And we should not forget that the younger groups, physicians and patients alike, have been weaned on a plethoric diet of advertising such as has never been seen or heard before. Even the young physicians will have to turn up the volume to get the attention of their own public.

So the problem of regulating physician advertising is only one facet of the new order. Society, in several of its forms, has determined that it is not physician advertising but the lack of it that may be detrimental to its health. Among our sins cited by the ayatollahs of consumerism is our denial to our patients of the benefits of open advertising. We are told that we have, with selfish intent, obstructed the patients' right to knowledge of available personnel, services, methods, options, and costs. Conversely, promotion of advertising would elevate patient care, increase healthy competition within the profession, extend the availability of all forms of health care, bring down costs, and straighten out just about all of the problems that confront the sociomedical world.

At the moment, the profession has sacrificed its outposts and is trying to firm up its defenses — it has acknowledged that the "directory" type of publicity is within acceptable limits. This "name, rank, and serial number" approach has been blessed by the medical Olympians (in fact was never disapproved but neither was it promoted) and has been put into effect in many areas. It is impossible to object to this procedure as long as it respects the fine line between information and advertising. But this small step acknowledges the opening of the door to increasing extension of publicity techniques. The comforts of total restriction are removed, and the profession must determine a new system of control which respects the principles of professional behavior without, in judicial view, interfering with public rights.

The objections of the profession to the more obvious and blatant forms of advertising have sound basis — the exploitation of patients, the denigration of ethical controls by the huckster mentality, the interference with the physician's role in managing his patient when referral or consultation is considered, for example. But now there is an added factor, the long arm of the bureaucratic enforcer. It is not enough to say that the patients are disserved by unrestrained advertising and to cite instances, convincing to us at least, in support. And it is not enough to say that medical advertising would never really reach the gross level. Already the advertisements of the California plastic surgery groups, though somewhat short of Madison Avenue's full treatment, are well beyond a dignified, professional form.

Now the threat of liability is imposed on organized medicine and its agents (such as this periodical) if it can be shown to a court's satisfaction that restraints of advertising from such organizations have interfered with the rights either of health care providers to publicize their services or the public to receive such information. Judicious handling by medical organizations might insure against adverse judgments but, as our clogged court calendars testify, such questions lead to numerous suits with expense in time, effort, and money regardless of the rectitude of the defense.

These comments to this point must be considered preamble. A thoroughly researched and carefully constructed bill, prepared by a committee of the Kansas Medical Society, under the chairmanship of a member carrying both medical and law degrees, was introduced during the recent legislative session. As frequently happens, the text and intent of the bill were gutted by amendments that would have removed all restraints on physician advertising and functioned in virtual opposition to the original intent. Fortunately, the altered bill was allowed to die. While this leaves advertising unrestricted by default for at least another year, this will permit a suitable restructuring of the bill and, in particular, an opportunity for members of the Kansas Medical Society to express their opinions as to the character of controls that should be made a part of the statutory control.

These remarks refer, of course, to the problem of advertising *by* physicians. Tangential to this is the matter of advertising in medical periodicals and aimed *at* physicians. Another expression of the shifting attitude toward physicians has been the charge from some quarters that such periodicals — and thus their readers, the physicians — are consequently under the nefarious influence of the advertisers, notably pharmaceutical firms. The truth of such charges is of little moment to the critics — it is the effect created by their pronouncements that they seek.

The Editorial Board has always assumed the responsibility for promoting and regulating the advertising that has appeared in the *Journal,* and its actions have apparently met with Society approval. A change in obligation, however, is produced by the prospect of physicians themselves being the advertisers. Since the Board's decisions on advertising

policy will obviously affect the tone of this publication (not to mention the legal implications), it is concerned that this policy reflect the attitude of the members of the KMS. To this end, we strongly urge our component groups as well as individual members to offer their opinions and desires. — *D.E.G.*

And another thing . . .

(Editor's Note: The following report by Warren H. Pearse, Executive Director of the American College of Obstetricians and Gynecologists, appeared in the February Newsletter of that organization. With Dr. Pearse's permission, we are submitting it, as he did, without further comment.)

Second opinion needs second opinion

The Health Care Financing Administration (HCFA) of the Department of Health, Education and Welfare (HEW) established a toll free number to be used by the U.S. public, so that people might be directed to a surgeon who would provide a second opinion after a surgical procedure had been advised. The program was directed particularly at care funded by Medicare and Medicaid. This service was publicized by brochures mailed to Medicare and many Medicaid recipients, and by briefings and news releases. Recent reports indicate that a new campaign to urge use of this federal service will be undertaken early in 1980.

Without editing or comment, an actual experience with this service is reported.

1. The published toll free number (800-325-6400) was called at 2:00 p.m. on a Wednesday afternoon. An answering recording indicated the number had been changed to 800-331-1000.

2. Dialing the new number led to busy signals twice. The third call reached an operator who requested the caller's zip code, state, and county. With this information, the operator provided an "EDS federal number" of 827-9570. Asked what EDS was, the operator responded that E was for education, but she did not know what the other initials were.

3. Dialing 827-9570 yielded a recording saying that the number had been changed to 635-6020.

4. Dialing 635-6020 yielded a recorded message saying "Thank you for calling Medicare. All telephone correspondents are busy, please wait." After a wait came another recording saying, "Lines are still busy, please hold on."

Finally a live voice answered and was asked for information about getting a second opinion for surgery recommended.

5. The federal operator asked questions
 —"Where do you live?"
 —"What kind of surgery?"
 —"Do you want another doctor from the same area?"

6. With this information, the federal operator said, "Hold on." The line remained open and the caller waited more than 10 minutes. The operator never returned.

Sunscreens

(Continued from page 251)

Answers

1. e
2. d
3. b
4. c
5. d

Suggested Readings

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Ka M P A C
KANSAS MEDICAL POLITICAL ACTION COMMITTEE

1300 TOPEKA AVE. • TOPEKA, KANSAS 66612

Dear Doctor:

Many physicians are becoming increasingly aware that federal bureaucracy tends to increase the importance of paperwork and decrease the importance of patients as individuals.

The physician's time is depleted by the demands of answering computer letters, signing computer-typed orders from nursing homes, and traveling about to deliver written instructions that will not be accepted by telephone.

This is not the type of medicine physicians have been trained to practice; it is not the type of medicine they want to practice. KaMPAC is working hard to keep medicine in the free enterprise system, which serves the patient best and at the least cost.

Your contribution and support are needed for KaMPAC to reach its goals. Please respond generously.

Sincerely,

Ronald Davis, M.D.
Chairman

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Human Protein Requirements

(Continued from page 230)

attenuate nitrogen loss and achieve nitrogen equilibrium.[17] High-quality protein (e.g., fish or fowl) is administered at 1.2-1.4 g/kg ideal body weight along with vitamins and trace elements. This protein intake cannot be substituted by nonprotein calories and still produce the same nitrogen balance. Protein clearly is essential to preserving body cell mass during hypocaloric feeding and severe calorie-restricted diets for treatment of obesity. While low-biological proteins may be capable of maintaining nitrogen balance when given in large amounts,[18] they have not been adequately tested as a protein source for unsupervised diets.

As of June, 1978, 58 deaths had occurred that were associated with the use of very low calorie, protein diets.[19] Sixteen of these deaths were due to development of cardiac arrhythmias and arrest in obese, but otherwise healthy, women who were reducing their body weight by strict adherence to a fasting regimen supplemented daily with small (3-5 oz) quantities of predigested liquid protein products. The etiology of these cardiac arrhythmias could not be determined by investigators from the Food and Drug Administration (FDA), Center for Disease Control or an *Ad Hoc* committee of scientific experts convened by the FDA. While there is support for the hypothesis that these deaths were due to severe caloric restriction and starvation, the most likely explanation is that deficiencies of certain essential minerals (i.e., potassium, magnesium, and phosphate) developed. Seven of these 16 deaths occurred during carbohydrate refeeding, a period in which significant metabolic changes from the starvation state occur (i.e., increased sodium and water retention, increased insulin production, and altered intracellular enzyme activity and breakdown rates of muscle and adipose tissue).

Stress, Injury and Infection

Protein requirements may be greatly increased during episodes of stress, infection, and injury. Alterations in the body protein metabolism are generally aimed at mobilizing skeletal muscle proteins to ensure an adequate supply of substrates for energy production and protein biosynthesis. This catabolism of muscle proteins is triggered by hormonal changes, such as increased catecholamine and glucocorticoid secretion into the bloodstream, resulting in an overall negative balance.[20] The degree of nitrogen loss is dependent upon the severity of infection or injury; obligatory losses may approach the equivalent of 0.9 g protein/kg/day.[21] Thus, prolonged infection and injury without adequate nutritional support will lead to protein malnutrition, as has been observed in hospitalized adult patients.[22] Basic oral anabolic diets for depleted patients have been described.[23] The protein-nitrogen content of these diets should be considered independent of calorie content. Nitrogen : calorie ratios are not instructive in designing dietary therapy. At a caloric intake of 45Kcal/kg/day, the desirable protein intake would be 1.8 g/kg/day to allow for optimal nitrogen utilization. At these levels, the ratio of nitrogen to calories is approximately 1 : 150. Special considerations for patients with liver or renal disease must be taken into account in terms of protein requirements.[23] During acute phase of injury, nitrogen intake may need to be further increased without an increase in nonprotein calories.

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Manic-Depressive Illness

PHYSICIANS are misdiagnosing nearly a fourth of all cases of manic-depressive illness, according to a Public Health Service study.

Between a third and a half of persons with this bipolar disorder are not receiving treatment; and nearly a fifth of those who are in treatment for the disorder don't have it, the study found.

If the diagnostic skills of physicians could be improved to levels of accuracy that are currently achievable, and optimal treatment could be provided to all who suffer from manic-depressive illness, hundreds of millions of dollars could be saved annually, states the recently released report.

The study employed teams of NIMH-selected experts on epidemiology, diagnosis, treatment, and economics to evaluate the state of knowledge and practice regarding bipolar disorder.

Some 34,000 cases of manic-depressive illness in 1976 would have accounted for $1 billion in economic losses in the U. S. that year had they gone untreated, the study estimated. The $222 million actually spent on treatment cut these potential losses to $441 million.

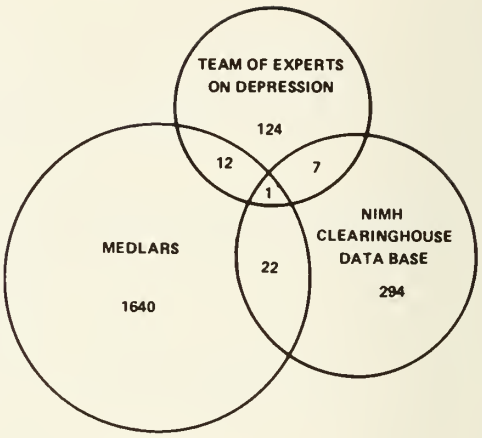
However, the incidence of misdiagnosis — now nearly one-fourth of all cases — could be cut in half if mental health specialists and primary practitioners in the community were better informed the report suggests.

Primary practitioners currently miss 28 per cent of manic-depressive cases they see and incorrectly label nine per cent of the 22 per cent of cases of the disorder they now identify, the study found.

Specialists, who detect 67 per cent of the cases, miss 15 per cent and wrongly label 10 per cent. Research clinicians miss 10 per cent, and wrongly label six per cent.

The report calls for educational efforts to improve the sensitivity of care givers to the problem. It recommends for physicians and other health professionals history taking, differential diagnosis, use of chemotherapy, monitoring blood lithium levels, and avoidance of inappropriate treatments, such as psychoanalysis for manic patients.

The expert team thought $20 million could be spent productively in the next five years researching bipolar disorder, and recommended that $16 million be allocated in light of research budget priorities.



Above: the number of journal citations on manic-depressive illness contained in each of three sources of information studied shows few citations shared in common. The communications gap could contribute to errors in diagnosis and treatment, according to an NIMH psychiatrist.

The study on bipolar disorder was one of three conducted under contract for the Assistant Secretary for Health between 1977 and 1979 as part of the Medical Practice Information Demonstration Project. The project summarized the most authoritative information available on selected health problems pertinent to quality assurance, medical education, and policy decision-making. Other disorders analyzed were malignant melanoma and rheumatic heart disease.

Dr. Robert Hirschfeld, the NIMH psychiatrist who served as project advisor for the study, noted that three major sources of information analyzed — the expert team; the NIMH clearinghouse's data base; and MEDLARS, a general medical data base — contained few journal citations in common. This lack of overlap points to a need for improved communication about bipolar disorder within the health care field, said Hirschfeld.

From *Alcohol, Drug Abuse, and Mental Health Administration News* 5:15, 1979.

FDA REGULATIONS DELAY DRUGS

Overly stringent Food and Drug Administration regulations and delay are unduly retarding approval of new drugs and discouraging investigators from developing them, the AMA has told Congress.

"We do not believe at this time the benefits derived from FDA's current safety and efficacy procedures and conservatism exceed the detriments that are caused by the lack of availability of new drugs in the U. S." said Ray Gifford, M.D., a member of the AMA's Council on Drugs and head of the Department of Hypertension and Nephrology at the Cleveland Clinic.

Dr. Gifford told a House Science Subcommittee that in recent years physicians have noticed "a disturbing trend that drugs ultimately approved in this country as major treatment break-throughs have been available in other countries for significant periods of time prior to U. S. marketing."

A complete list of drugs that were marketed earlier in foreign countries include important drugs that could have made significant improvements in therapeutics had they been available earlier in the United States, Dr. Gifford said.

"Two of the most potent diuretics we how have, ethacrynic acid and furosemide, were marketed in Europe fully two years before American physicians had access to them," Dr. Gifford said. "They are the only diuretics that are effective for patients with impairment of kidney function."

He noted that in 1963, he published the results of an investigation of a new drug, bethanidine, confirming the findings of European and Australian investigators that it was a potent and useful drug. Bethanidine was approved in the United Kingdom in 1963. Subsequently, it has become available in every other country in the world (including Canada), except the United States! "This has led to the situation where I can prescribe bethanidine for my Canadian patients and not for my American patients."

Dr. Gifford described, as an example, the use of sodium nitroprusside for hypertensive crisis which appeared more than 20 years ago but was not accepted for marketing by the FDA until 1974.

"There must be something wrong with our drug regulatory system when a drug that is recognized by authorities in the field as unique and lifesaving is not made available to the practicing physician for 20 years after the first description of its potential value," he said.

The AMA witness said it was found that in approving a drug, 25 times as much paperwork was required in the U. S. as compared to England, and it took twice as long to process the application here. He urged changes in the drug laws that would provide for the HEW Secretary to allow the limited marketing of "breakthrough" drugs without the receipt of full efficacy data and that otherwise would speed approval.

PILL BOTTLE SAFETY CAPS

The dialogue on the problems of adults in trying to open the child-resistant safety caps on pill bottles continues. In a recent issue of *JAMA*, a California pharmacist writes that "pharmacists should offer their elderly patients the option of having a non-child-resistant container" because many elderly persons have difficulty in removing the safety caps.

Says Gail S. Geiger, Pharm.D., of Huntington Memorial Hospital, Pasadena: "Elderly patients have related many instances to me describing their dilemma in trying to open their prescription containers and the variety of instruments they have used in the process: pliers, hammer, screwdriver, hand-operated can openers and feet. One patient claimed that stomping on his bottle worked to free its contents.

"Another patient stated that her 7-year-old granddaughter could open her bottle when she, herself, could not."

Elderly patients should be routinely screened by druggists to determine whether they can manage the child-resistant safety cap. If they have a problem, a simpler cap may be substituted, Dr. Geiger says.

In another communication, Matilda S. McIntire, M.D., and Carol R. Angle, M.D., of the American Association of Poison Control Centers, Omaha, also urge that health professionals caring for the elderly demonstrate the container's proper use, and test the patient's ability to open the cap.

The safety caps have brought a marked reduction in incidence of accidental poisoning of small children from pills and other medicines. But the safety caps also have been such a problem to some adults that they give up and neglect taking their medications.

NEW APPROACH TO MEETINGS

The American Medical Association in 1980 will launch a new concept in medical meetings for doctors — the Theme Meeting.

In collaboration with many of the medical specialty societies, the AMA will begin a series of meetings each centering on one clinical theme organized around a disease, an organ system or a group of related treatments.

First theme meeting on coronary artery disease was held April 10-13, at Los Angeles. Second meeting will feature clinical drug therapies, at Kansas City, Sept. 27-30.

Organized by the AMA's Council on Continuing Physician Education, successor to the former Council on Scientific Assembly, the meetings offer

new and clinically relevant information in a given medical area for both the specialist and the primary-care physician. Programs will be targeted for the non-specialist, but will provide advanced, sophisticated information that will cut across specialty lines. Much of the meeting time will be devoted to workshops where the physicians will be given hands-on demonstrations and one-on-one or small-group discussion of specific problems.

Cooperating in the coronary heart disease meeting in April in California were the American College of Cardiology, American College of Chest Physicians, American Association for Thoracic Surgery, American Society of Clinical Pathologists, American College of Physicians, and American Society for Clinical Pharmacology and Therapeutics.

The meeting offered a comprehensive review of coronary heart disease, allowing doctors to study how to evaluate risk of heart disease in their patients, stress testing in office practice, rehabilitation after heart attack, management of angina, prevention of heart disease, how to read the electrocardiogram, the role of surgery in heart disease, and other aspects of the problem.

BREAST CANCER

New research studies have confirmed the effectiveness of a drug that has proved useful in treating a breast cancer that has spread to other areas of the body.

The drug is tamoxifen citrate. Trade name is Nolvadex. The drug has been available to American physicians for the past year.

The *Journal of the American Medical Association* in July published a report of tests of tamoxifen on 50 patients at M. D. Anderson Hospital and Tumor Institute, Houston. These were breast cancer patients in whom conventional endocrine therapy and combination chemotherapy (drugs) had failed, says Sewa S. Legha, M.D. In all of them the cancer had spread.

Tamoxifen achieved a significant palliation of disease in 50 per cent of patients with far-advanced breast cancer that had failed to respond to conventional treatments, Dr. Legha says.

"Since this result was obtained at the cost of practically no side effects, the results are indeed remarkable," he declares.

LETTER FROM AN EDITOR

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Which I will do with haste,
When you send another copy —
The first has been misplaced.

George Banks, M.D.
Tustin, California

Dr. Banks is Director, Ethical Division — ICN Pharmaceuticals, and a consultant with Research Health Sciences, Inc. He has edited medical publications for the pharmaceutical industry, and his light verse has appeared in the *New England Journal of Medicine*.

Reprinted from *Medical Communications*, Vol. 7, No. 4.

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PATIENT WARNING: Should not be used in the presence of undiagnosed abdominal pain. Frequent or prolonged use without the direction of a physician is not recommended. Such use may lead to laxative dependence.

DIRECTIONS FOR USE—ADULTS: Before breakfast and after the evening meal, one to two rounded teaspoonfuls of Perdiem™ granules should be placed in the mouth and swallowed with a full glass of warm or cold beverage. Perdiem™ granules should not be chewed. After Perdiem™ takes effect (usually after 24 hours, but possibly not before 36-48 hours), reduce the morning and evening doses to one rounded teaspoonful. Subsequent doses should be adjusted after adequate laxation is obtained.

IN OBSTINATE CASES: Perdiem™ may be taken more frequently, up to two rounded teaspoonfuls every six hours.

FOR PATIENTS HABITUATED TO STRONG PURGATIVES: Two rounded teaspoonfuls of Perdiem™ in the morning and evening may be required along with half the usual dose of the purgative being used. The purgative should be discontinued as soon as possible and the dosage of Perdiem™ granules reduced when and if bowel tone shows lessened laxative dependence.

FOR COLOSTOMY PATIENTS: To ensure formed stools, give one to two rounded teaspoonfuls of Perdiem™ in the evening with warm liquid.

DURING PREGNANCY: Give one to two rounded teaspoonfuls each evening.

FOR CLINICAL REGULATION: For patients confined to bed, for those of inactive habits, and in the presence of cardiovascular disease where straining must be avoided, one rounded teaspoonful of Perdiem™ taken once or twice daily will provide regular bowel habits. Take with a full glass of water or beverage.

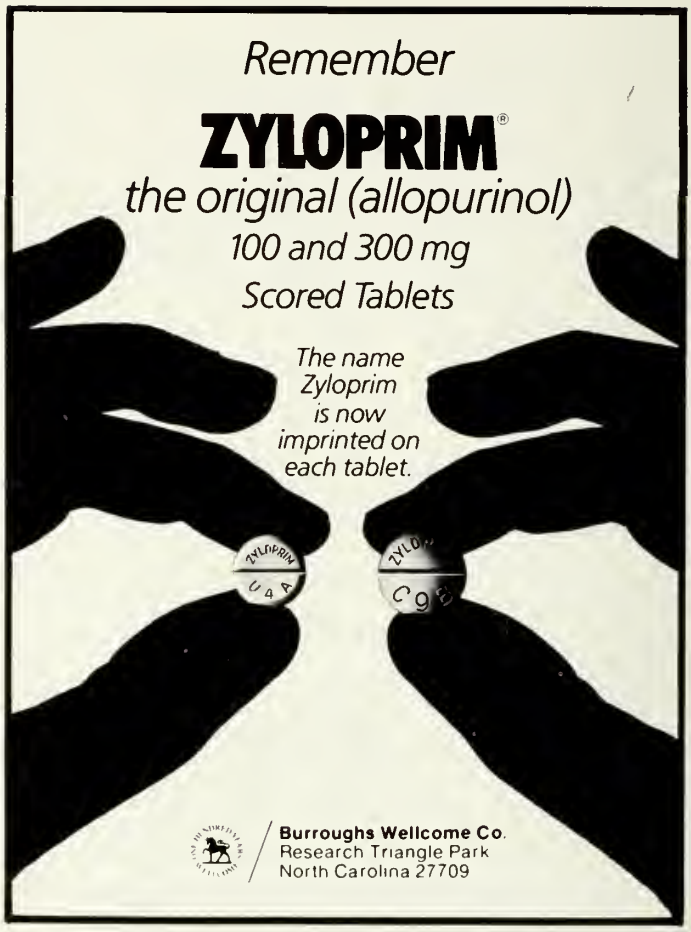
FOR CHILDREN: From age 7—11 years, give one rounded teaspoonful one to two times daily. From age 12 and older, give adult dosage.

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Human Protein Requirements

JOHN D. PALOMBO, M.L.S., M.S. *and*
GEORGE L. BLACKBURN, M.D., Ph.D.

NEXT TO WATER, protein is frequently considered the quintessential nutrient, since it is the most essential constituent of cell metabolic components. The protein content of the adult human body is approximately 12 kilograms, half of which is contained in the body cell mass. The predominant protein mass of the body that requires dietary protein for replenishment of indispensable amino acids is the lean body mass (i.e., fat-free muscle, viscera, and connective tissue). The protein content of the human body is constantly undergoing synthesis and breakdown. The rates and directions of these processes in the various tissues are dependent upon many factors, including the age and physical condition of the subject, the dietary intake of calories and protein, and physiological and pathological occurrences, i.e., starvation, infection, stress, and disease.

The liver and muscle proteins, which constitute a major part of the body cell mass, are synthesized from dietary amino acids and amino acids provided from tissue protein breakdown. Surplus amino acids are used to produce carbon skeleton molecules or are broken down into carbon dioxide, water, energy and nitrogen-based radicals for excretion or utilization in other compounds. In the liver, the amino group is converted to urea and subsequently excreted in the urine.

Protein nitrogen is lost continuously through the urine, feces and skin, as well as through hair and nail losses. The daily protein supply for tissue protein synthesis must, therefore, be continuously replenished.

Dietary requirements for protein nitrogen include a need for certain amino acids that cannot be synthesized in the body. These are called essential or indispensable amino acids (EAA). For humans there are eight essential amino acids; however, infants and stressed older humans require an additional amino acid. Nonessential amino acids can be synthesized in the body.

The nutritional value of a specific dietary protein depends on the relative quantities of all the amino acids it contains. Dietary proteins that contain all the essential amino acids in sufficient amounts and proportions to each other are considered to be *complete* or *high-quality proteins*.[1] These proteins provide all the amino acids in the proportions needed by the body for growth and tissue maintenance. Proteins from animal sources (meat, milk, eggs) are examples of high-quality proteins.

Proteins from individual vegetables and grains tend to have *incomplete* patterns of essential amino acids, i.e., they are generally deficient in one or more essential amino acids and are, therefore, of lower quality. Although individual vegetable and grain proteins are of lower quality, *mixtures* of these proteins can result in a total protein intake of higher nutritional quality than the components themselves.[1] This occurs when the deficient or limiting essential amino acid in one food source in an individual's diet is offset by the presence of that amino acid consumed in another food. For example, wheat products that are deficient in lysine have moderate quantities of methionine; legumes, however, have an inverse ratio of these essential amino acids. When these two protein sources are consumed in the same meal, the proportions of lysine and methionine will be balanced out, yielding a meal with a higher protein quality than either individual protein would yield. Thus, vegetarians who select a diet containing a wide variety of foods are able to maintain adequate protein synthesis and avoid protein deficiency.

Utilization of dietary amino acids for protein synthesis by the liver occurs during the absorption from the intestine. Both human and animal studies have demonstrated impairment of liver protein synthesis during this absorptive period if tryptophan is not consumed in the same meal with the other essential amino acids. The evidence is less clear in regard to impairment of the synthesis of human liver proteins as a result of a short term (i.e., less than 24 hrs) dietary limitation of any one of the other essential amino acids.[2]

It is important to realize that the ability of protein to meet the requirement of nitrogen balance is not only a function of biological value, but is also a function of the total amount of protein taken in. Thus, while 27-30 grams (g) of a vegetable protein mixture may not be adequate for nitrogen balance, feeding this same vegetable protein at a normal in-

From the Nutrition/Metabolism Laboratory, Cancer Research Institute, New England Deaconess Hospital/Harvard Medical School, Boston, Massachusetts.

take of 75 g/day can produce nitrogen balance and be as acceptable a protein source as "higher biological value" protein. Nitrogen balance is also dependent on other important variables: the availability of non-protein calories (particularly carbohydrate), age of a person, and severity of disease. A number of techniques are available to determine the protein quality of a given food and are described in detail elsewhere.[3]

Determination of Protein Requirements

Human protein requirements have been investigated intensively during the past 50 years using either the Factorial Method or the Nitrogen Balance Method.[4] Protein requirements vary from individual to individual according to age, diet, environmental factors, pathological conditions, etc.[4] For example, on a body weight basis, the amount (grams) of protein required per kilogram body weight (kg BW) decreases with age.[5] Furthermore, the proportion of dietary protein that must be supplied as essential amino acids decreases with age; while 32 per cent of total estimated protein requirements in children should be supplied as essential amino acids, adults require only 15 per cent as essential amino acids.[4] In other words, on a body weight basis, adults require 78 milligrams (mg) essential amino acids per kg BW daily, whereas children require 214 mg essential amino acids per kg BW.[5] Thus, dietary proteins appear to be used more efficiently by adults in meeting requirements than by children.

Stress, infection, and heat can cause increased nitrogen loss and increased protein requirements. Heavy work or intensive physical training exercises cause an increase in muscle mass.[6] Therefore, athletes in training may have a slight increase in protein requirements.

In the United States, these protein needs will undoubtedly be met by dietary means, since 97 per cent of healthy Americans consume much more protein than is required; the amount of extra dietary protein consumed may approach 40 to 50 g/day.[7]

Energy Intake and Protein Requirements

Standard estimates of protein requirements are valid only when energy needs have been met. When energy intake is inadequate, some dietary and tissue protein will be oxidized or converted to glucose in the liver to meet energy needs. When energy intake levels are marginal, protein needs are higher than during periods of satisfactory energy intake.[8] This may be due to relative changes in activities or concentrations of key enzymes responsible for degradation of EAA and an obligatory requirement for energy substrates from amino acids. Thus, efficiency of nitrogen utilization, which in turn affects protein requirements, is dependent upon the total calorie intake and energy requirements.[9]

Recommended Dietary Allowances

Since protein requirements for a given age, weight, and sex group vary widely according to the previously discussed factors, recommended protein intakes had to be set substantially higher than minimum needs in order to cover the needs of the greater majority of individuals within these groupings.

The Recommended Dietary Allowances (RDA) for protein in adults (23-50 years of age) are based on results from nitrogen balance studies which determined the average maintenance requirement for nitrogen as protein.[10] This amount, which is 0.47 g protein/kg BW, is increased 30 per cent to cover individual variability. The efficiency of protein utilization (75%) in the average U. S. diet is then considered; thus 0.8 g protein/kg/day (56 g for a 70 kg male, and 44 g for a 55 kg female) is derived as the daily protein allowance. Pregnant and lactating women have higher protein allowances — an extra 30 g and 20 g respectively. The 1973 Technical Report #522 by FAO/WHO on energy and protein requirements recommended lower intakes based on egg or milk protein: 0.57 and 0.52 g protein/kg for adult men and women, respectively.[3] This report further suggested that a correction for dietary protein quality might be necessary for certain age groups or individuals on certain diets. Therefore, a diet consisting primarily of low-quality proteins would necessitate increasing the recommended intake of protein to a new "safe" level. Problems in the interpretation and use of dietary standards have been reviewed elsewhere.[11]

Protein Intake in the United States

In the U. S., approximately 15-17 per cent of the total caloric intake is in the form of protein, which is equivalent to 90-100 g/day.[12] Since minimal protein needs for healthy adults are 35-40 g/day, and the RDA are 44-56 g, it can easily be understood why protein supplements are superfluous and are mainly an expensive source of energy.[1] The findings from various U. S. government nutrition surveys such as the Ten-State Nutrition Survey, the Health and Nutrition Examination Survey (HANES), and the new U. S. Department of Agriculture (USDA) Nationwide Household Food Consumption Survey indicate that the vast majority of Americans receive adequate dietary protein for body maintenance and good health, and that protein deficiency is a rare

(Continued on page 230)

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Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

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Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

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Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

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Full information is available on request.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability, insomnia associated with anxiety neuroses and transient situational disturbances; anx associated with depressive symptoms and as a treatment of symptoms of anxiety if such sy toms are a significant feature of functional or organic disorders, particularly gastrointestina cardiovascular.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by syst atic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucom

Warnings: Not recommended in primary depressive disorders or psychoses. As with all C acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, an diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with ba turates and alcohol have occurred following abrupt discontinuance of benzodiazepi (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Ad tion-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveilla when on benzodiazepines because of their predisposition to habituation and depender Withdrawal symptoms have also been reported following abrupt discontinuance of benz azepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid o sedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may resu symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and oc sional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazep has not been shown of significant benefit in treating gastrointestinal or cardiovascular com nent.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 yea 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum hur therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn wi 2 months of first observation. Clinical significance is unknown, but use of lorazepam for longed periods and in geriatric patients requires caution and frequent monitoring for sympt of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia, some have elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function t are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depres effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerge rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rab Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastrosch malformed skull and microphthalmia) were seen in drug-treated rabbits without relationshi dosage. Although all these anomalies were not present in the concurrent control group, have been reported to occur randomly in historical controls. At 40mg/kg and higher, there evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lo doses. Clinical significance of these findings is not known. However, increased risk of cong tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam meprobamate) during first trimester of pregnancy has been suggested in several stud Because use of these drugs is rarely a matter of urgency, use of lorazepam during this pe should almost always be avoided. Possibility that a woman of child-bearing potential may pregnant at institution of therapy should be considered. Advise patients if they become p nant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazep and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like o benzodiazepines. As a general rule, nursing should not be undertaken while on a drug si many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and gener disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxi patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9 weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, r sea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, function disturbance, various gastrointestinal symptoms and autonomic manifestations. I dence of sedation and unsteadiness increased with age. Small decreases in blood press have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple age may have been taken. Manifestations of overdosage include somnolence, confusion and co Induce vomiting and/or undertake gastric lavage followed by general supportive care, moni ing of vital signs and close observation. Hypotension, though unlikely, usually may be contro with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® (lorazepam) IV for Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dc gradually when needed, giving higher evening dose before increasi daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosa may vary from 1 to 10mg/day in divided doses. For elderly or deb tated, initially 1-2mg/day; insomnia due to anxiety or transient sitt tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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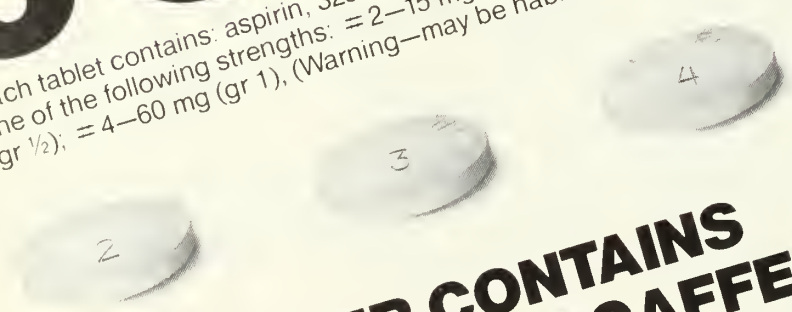
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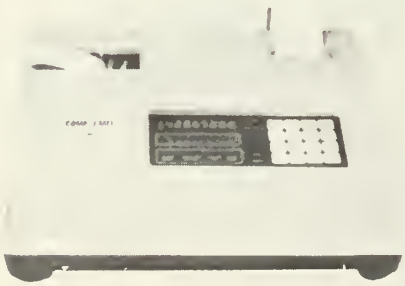
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Hyperparathyroidism

(Continued from page 244)

Cardiovascular manifestations of hypercalcemia include shortening of the Q-T segment of the EKG, hypertension, and vascular calcifications. In addition, an ocular change associated with hyperparathyroidism is band keratopathy.

In summary, the pathophysiology of hyperparathyroidism has been elucidated over the past 50 years. All tissues are affected by the consequences of elevated parathormone and resultant hypercalcemia. This produces a disease complex with protean manifestations and varied clinical presentations.

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Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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RESOLUTIONS

THE Journal OF THE Kansas Medical Society

JUNE
1980

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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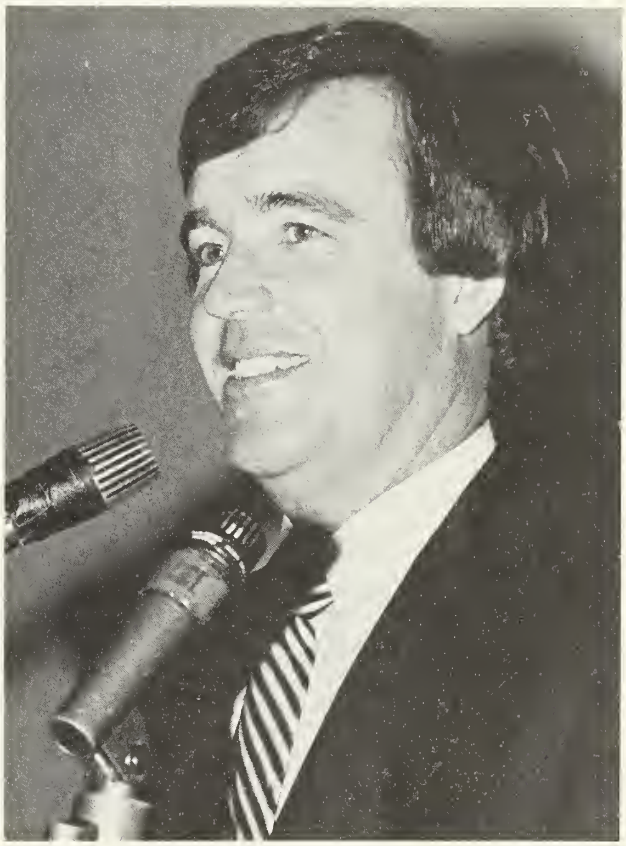
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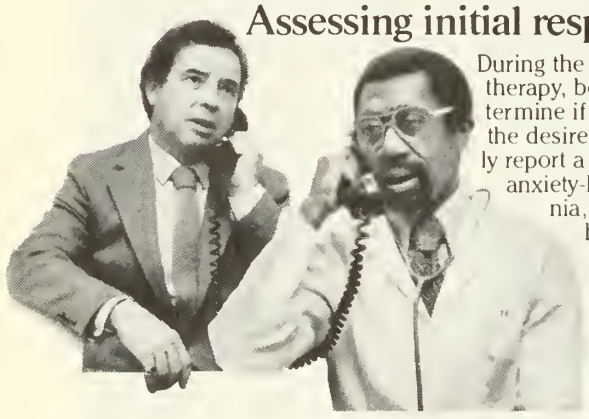


The Power and the Glory: The Auxiliary of the Kansas Medical Society presents a united front as another year ends and begins: (left to right) Caryl Bichlmeier, Phyllis Bigler, Diane Sanders, Meldon Laury, Betty Moore, Evelyn Huff, and Kathy Wedel.

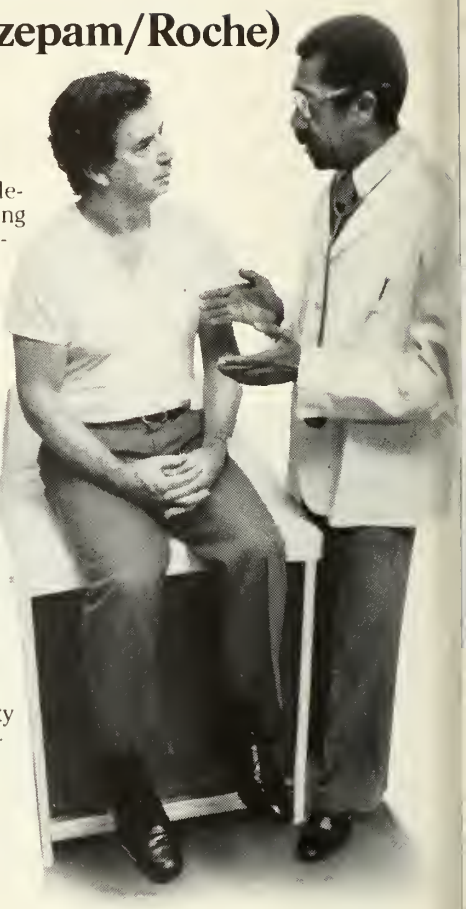
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Monitoring patient response to Valium® (diazepam/Roche)

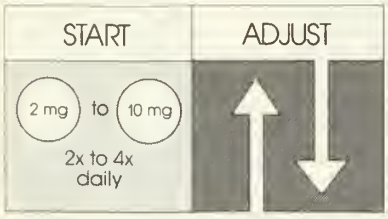
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During the first follow-up visit after initiating therapy, both physician and patient should determine if Valium (diazepam/Roche) is having the desired effect. Most patients will promptly report a feeling of relaxation and relief of anxiety-linked symptoms such as insomnia, headaches, palpitations and hyperventilation. You will probably observe that the patient is calmer and more relaxed. If, however, patient response does not measure up to expectations, a reevaluation of the patient's profile with modification of the dosage regimen should be considered.



Making dosage adjustments



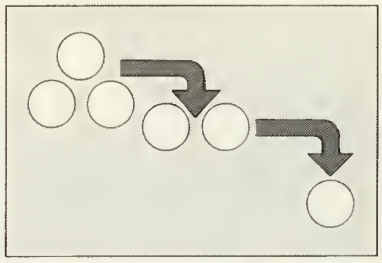
With any psychoactive medication it is good medical practice to initiate therapy at base dosage levels and titrate to the patient's needs. With Valium, experience has shown that 5 mg t.i.d. is usually sufficient although some patients with severe or persistent anxiety may require higher dosages initially. In geriatric or debilitated patients, the recommended dosage is 2 to 2½ mg once or twice daily. When anxiety fluctuates, as is common with most patients, the dosage may be adjusted as needed during the course of therapy; three strengths in scored tablets give you unmatched flexibility and simplicity in individualizing dosage.

Evaluating progress toward therapeutic goals

SET GOALS						
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

At the beginning of therapy it is now common practice for both physician and patient to establish treatment goals and to estimate the amount of time needed to achieve them. Then the patient knows what to expect and when to expect it. Some physicians find that compiling a checklist of presenting symptoms and complaints is useful for assessing the patient's response from visit to visit. In this way, progress toward attainment of the therapeutic goal is reviewed at regular intervals. As patients feel their symptoms abate and begin to develop insight into the sources of their anxiety and psychic tension, the checklist can be expected to dwindle.

Discontinuing pharmacologic intervention



When you decide to discontinue therapy, tapering dosage is good medical practice. Although rarely necessary after short-term treatment with Valium, gradual dosage reduction is advisable for patients who have been on extended therapy. This gradual discontinuance should preclude either recurrence of pretreatment symptoms or development of untoward side effects. Symptoms of withdrawal have almost always been associated with abrupt discontinuance of therapy of higher dosages taken continuously over long periods of time.

2-mg, 5-mg, 10-mg scored tablets

Valium® (IV)
diazepam/Roche

An Important Adjunct to Your Treatment Program for Excessive Anxiety

ROCHE®

See the following page for a summary of product information.

Valium®(diazepam/Roche) ℞

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

ROCHE®
Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.



Burdick introduces the 38-second electrocardiogram.

In fact, only 37.4 seconds to record the complete 12-lead electrocardiogram. It's the new EK-8, Burdick's first fully automatic single-channel electrocardiograph.

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Burdick card and folder-format mounts—fast, efficient way to mount and file ECGs

GOETZE-NIEMER



WHEN ANXIETY AND TENSION MAGNIFY PAIN

IN MUSCULOSKELETAL DISEASE*

A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC® (IV)

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective, for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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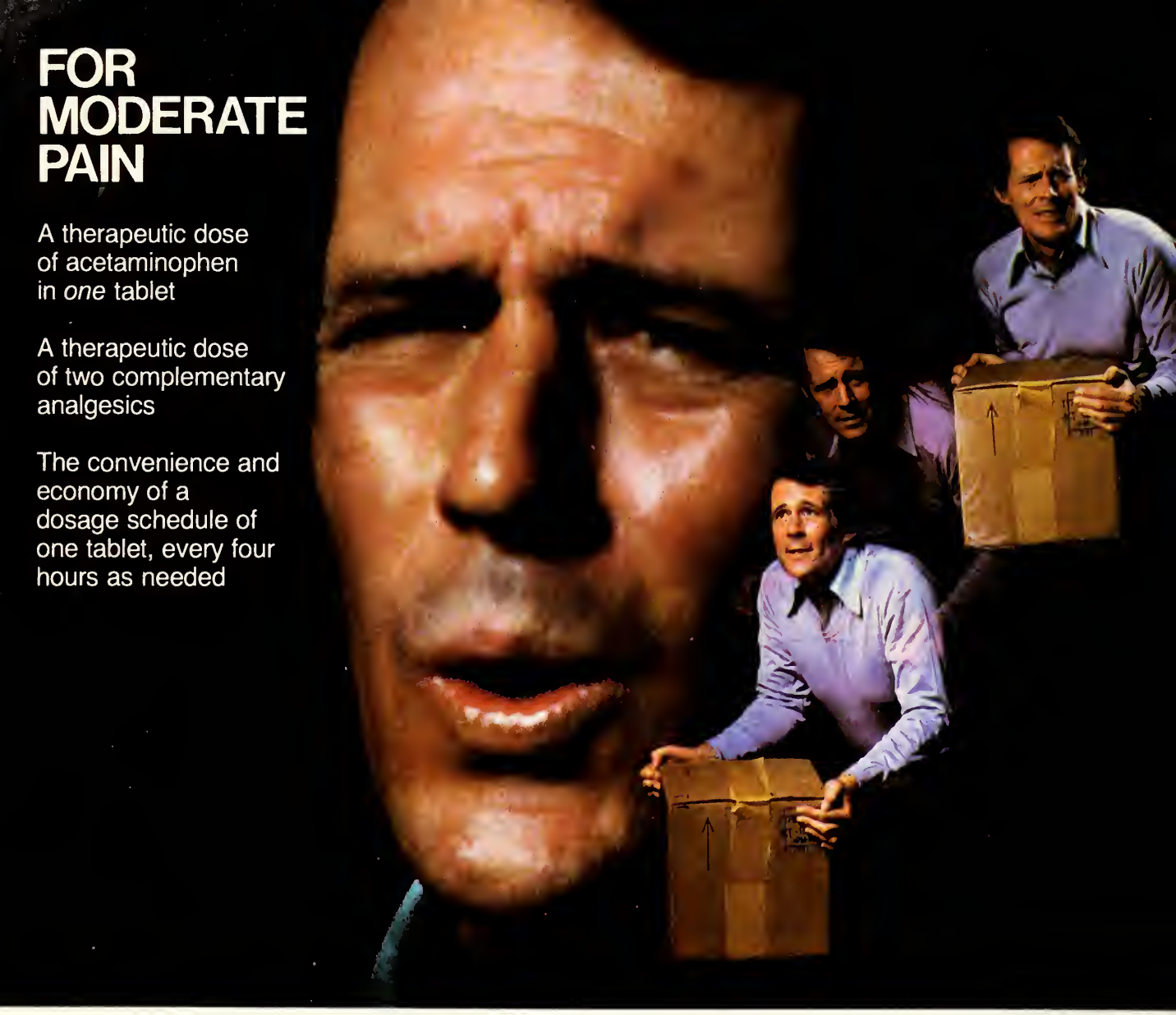
TM

FOR MODERATE PAIN

A therapeutic dose of acetaminophen in *one* tablet

A therapeutic dose of two complementary analgesics

The convenience and economy of a dosage schedule of one tablet, every four hours as needed



WHY NOT WYGESIC® C IV

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE. Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Manegement of Overdosege**)

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine s although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly

USAGE IN PREGNANCY: Sate use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY. Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients teking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. (see **Warnings**) Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine

MANAGEMENT OF OVERDOSAGE: SYMPTOMS. The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal

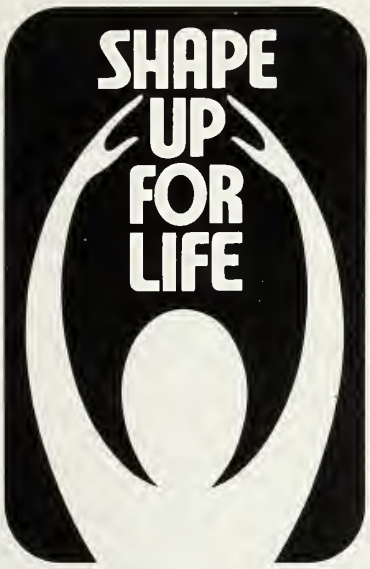
TREATMENT: Primary attention should be given to the reestablishment of adequete respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An eppropriate dose of one of these antagonists should be administered, preferebly IV, simultaneously with efforts at respiretory resuscitation and the antagonist should be repeated as necessary until the patient s condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used beceuse of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information. (JAMA 237 2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended

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American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

AUXILIARY NEWS

An Open Letter to Kansas Physicians

The Auxiliary enjoyed a very successful convention in Overland Park, thanks to Kathy Wedel, immediate past president, and the co-hostesses from the Johnson and Wyandotte County Auxiliaries.

The "Shape Up for Life" program was implemented by Kathy Wedel with an aerobics exercise participation group. Some 20 auxilians donned warm-up suits, and for two hours found that "it's fun to exercise with music."

The auction on Friday night was a first for Kansas. We heard so many good comments from the physicians and their spouses that we are already planning another one for next year's convention. Caryl Bichlmeier worked hard to provide many beautiful, useful, fun, and collectible items for sale. From the proceeds, AMAERF received $3,479, and Project Concern — in honor of Dr. and Mrs. Wilbur Cauble — received $1,235. We thank all the auxiliaries and individuals who donated items and those who participated for a most successful evening.

The AMAERF check presented to the University of Kansas Medical Center was the second highest per capita in the nation given to a medical school. The amount was $24,681.19. The Wichita branch received $3,126.26.

We are most pleased that the President of the AMA Auxiliary, Mrs. Ben Johnson, Jr., honored us with her presence throughout the convention. She was guest speaker at our Saturday luncheon. We are looking forward to attending National Convention in Chicago in July.

Preparations are under way for the Auxiliary summer board meeting on June 13 in Overland Park. The business meeting will begin at 9 AM, followed by an officers training session. The state chairmen will present their programs for the year and the county presidents will explain their projects. We urge all county presidents, presidents-elect, and committee chairmen to attend.

The "Shape Up For Life" logo shown on this page represents one of our ongoing programs for this year. Food for Fitness was stressed last year, and the focus will be on physical fitness in 1980-81. Posters are available for your office as well as iron-ons for joggers' shirts. Materials and information are available from AMA Auxiliary, 535 N. Dearborn, Chicago, IL 60610.

Sincerely,
Evelyn Huff
President
Kansas Medical Society Auxiliary

Personalities

Rex R. Fischer, Manhattan, has been re-elected Chairman of the Board of Directors of Blue Shield of Kansas.

John D. Robinson, Kansas City, has returned from Quito, Ecuador, where he served for one week as consultant to the Ecuadorian Society of Anesthesiologists. He lectured and held demonstrations primarily in cardiac anesthesia and invasive monitoring techniques. He was sponsored by the World Health Organization.

William G. Eckert, Wichita, is editor of the recently released "Introduction to Forensic Sciences," the first introductory textbook in forensic sciences published and used in America.

Timothy M. Scanlan, Wichita, has been elected president of the Kansas Jaycees.

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LIBRARIES

YOUR LIBRARIAN WILL BE

HAPPY TO ASSIST YOU

• provides effective symptomatic relief
• *b.i.d.* dosage simplifies therapy
• scored tablet for dosage flexibility

OPTIMINE®
azatadine maleate, 1 mg. tablets

CONTRAINDICATIONS Use in Newborn or Premature Infants. This drug should not be used in newborn or premature infants.

Use in Nursing Mothers. Because of the higher risk of antihistamines for infants generally and for newborns and prematures in particular, antihistamine therapy is contraindicated in nursing mothers.

Use in Lower Respiratory Disease. Antihistamines should NOT be used to treat lower respiratory tract symptoms including asthma

Antihistamines are also contraindicated in the following conditions: hypersensitivity to azatadine maleate and other antihistamines of similar chemical structure, monoamine oxidase inhibitor therapy (See DRUG INTERACTIONS Section)

WARNINGS Antihistamines should be used with considerable caution in patients with: narrow angle glaucoma, stenosing peptic ulcer, pyloroduodenal obstruction, symptomatic prostatic hypertrophy, bladder neck obstruction

Use in Children. In infants and children especially, antihistamines in overdosage may cause hallucinations, convulsions, or death

As in adults, antihistamines may diminish mental alertness in children. In the young child, particularly, they may produce excitation

OPTIMINE TABLETS ARE NOT INTENDED FOR USE IN CHILDREN UNDER 12 YEARS OF AGE

Use in Pregnancy. Experience with this drug in pregnant women is inadequate to determine whether there exists a potential for harm to the developing fetus.

Use with CNS Depressants. Azatadine maleate has additive effects with alcohol and other CNS depressants (hypnotics, sedatives, tranquilizers, etc.)

Use in Activities Requiring Mental Alertness. Patients should be warned about engaging in activities requiring mental alertness, such as driving a car or operating appliances, machinery, etc

Use in the Elderly (approximately 60 years or older): Antihistamines are more likely to cause dizziness, sedation, and hypotension in elderly patients

PRECAUTIONS Azatadine maleate has an atropine-like action and, therefore, should be used with caution in patients with: a history of bronchial asthma, increased intraocular pressure, hyperthyroidism, cardiovascular disease, hypertension

DRUG INTERACTIONS MAO inhibitors prolong and intensify the anticholinergic (drying) effects of antihistamines.

ADVERSE REACTIONS The most frequent adverse reactions are underlined:

General: Urticaria, drug rash, anaphylactic shock, photosensitivity, excessive perspiration, chills, dryness of mouth, nose, and throat

Cardiovascular System: Hypotension, headache, palpitations, tachycardia, extrasystoles

Hematologic System: Hemolytic anemia, thrombocytopenia, agranulocytosis

Nervous System: Sedation, sleepiness, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesias, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis, hysteria, neuritis, convulsions

Gastrointestinal System: Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation

Genitourinary System: Urinary frequency, difficult urination, urinary retention, early menses

Respiratory System: Thickening of bronchial secretions, tightness of chest and wheezing, nasal stuffiness

OVERDOSAGE Antihistamine overdosage reactions may vary from central nervous system depression to stimulation. Stimulation is particularly likely in children. Atropine-like signs and symptoms (dry mouth, fixed, dilated pupils, flushing, and gastrointestinal symptoms) may also occur

If vomiting has not occurred spontaneously, the patient should be induced to vomit. This is best done by having him drink a glass of water or milk after which he should be made to gag. Precautions against aspiration must be taken, especially in infants and children

If vomiting is unsuccessful, gastric lavage is indicated within three hours after ingestion and even later if large amounts of milk or cream were given beforehand. Isotonic and ½ isotonic saline is the lavage solution of choice

Saline cathartics, such as milk of magnesia, draw water into the bowel by osmosis and therefore are valuable for their action in rapid dilution of bowel content

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Council Meeting

Report of Meeting Held May 4, 1980

A meeting of the Council was held on Sunday, May 4, 1980, at Glenwood Manor Motor Hotel, Overland Park, following adjournment of the second session of the House of Delegates. Present were Drs. Phillip A. Godwin, President, Lawrence; Louis G. Allen, Shawnee Mission; Kenneth M. Boese, Manhattan; Richard L. Brownrigg, Dodge City; Clair C. Conard, Dodge City; Jack R. Cooper, Shawnee Mission; Louis M. Culp, Kansas City; Herbert Fransen, Newton; Jimmie A. Gleason, Topeka; Donald D. Goering, Coldwater; Herman W. Hiesterman, Quniter; Robert W. Hughes, Lawrence; Robert F. Moore, Caney; John R. Neuenschwander, Hoxie; Ivan E. Rhodes, Wichita; Alex Scott, Junction City; Marvin D. Snowbarger, Emporia; William C. Swisher, Wichita; Max E. Teare, Garden City; William K. Walker, Sedan; Roger D. Warren, Hanover; Wallace N. Weber, Hays; Kermit G. Wedel, Minneapolis; Emerson D. Yoder, Denton; and John O. Yulich, Kansas City.

Also present were Eleanor Bell, Val Braun, Jerry Slaughter, and Gary Caruthers.

David E. Gray, M.D., was unanimously reappointed editor of *The Journal of the Kansas Medical Society;* and John A. Segerson, M.D., and M. Martin Halley, M.D., were reappointed to serve additional three-year terms on the Editorial Board.

Two items of business referred from the House of Delegates were addressed, and both are to be further considered by committees appointed by Dr. Godwin. These two items are: (1) Resolution 80-22 relating to possible definition of Practice of Medicine; and (2) Resolution 80-31 relating to autopsies.

The current policy regarding interest charges on accounts for physicians' services was reaffirmed. This policy reads as follows:

> It is not in the best interest of the public or the profession to charge interest on an unpaid bill or note, or to charge a penalty on fees for professional services not paid within a prescribed period of time, or to charge a patient a flat collection fee if it becomes necessary to refer the account to an agency for collection.
>
> It is not improper, however, for a physician to add a service charge, equal to the actual administrative cost of rebilling, on accounts not paid within a reasonable time. The patient must be notified in advance of the existence of this practice.

Dr. Godwin requested that arrangements be made for his visits to the Council districts for summer and autumn insofar as possible.

Jerry Slaughter reported on actions of the 1980 session of the Legislature, with particular attention to the Open Records Act.

It was announced that a meeting has been scheduled for Thursday, May 8, with Dr. Godwin; Senator Ross Doyen; Robert Harder, Secretary of SRS; and representatives of other health provider groups to discuss Title XIX reimbursement.

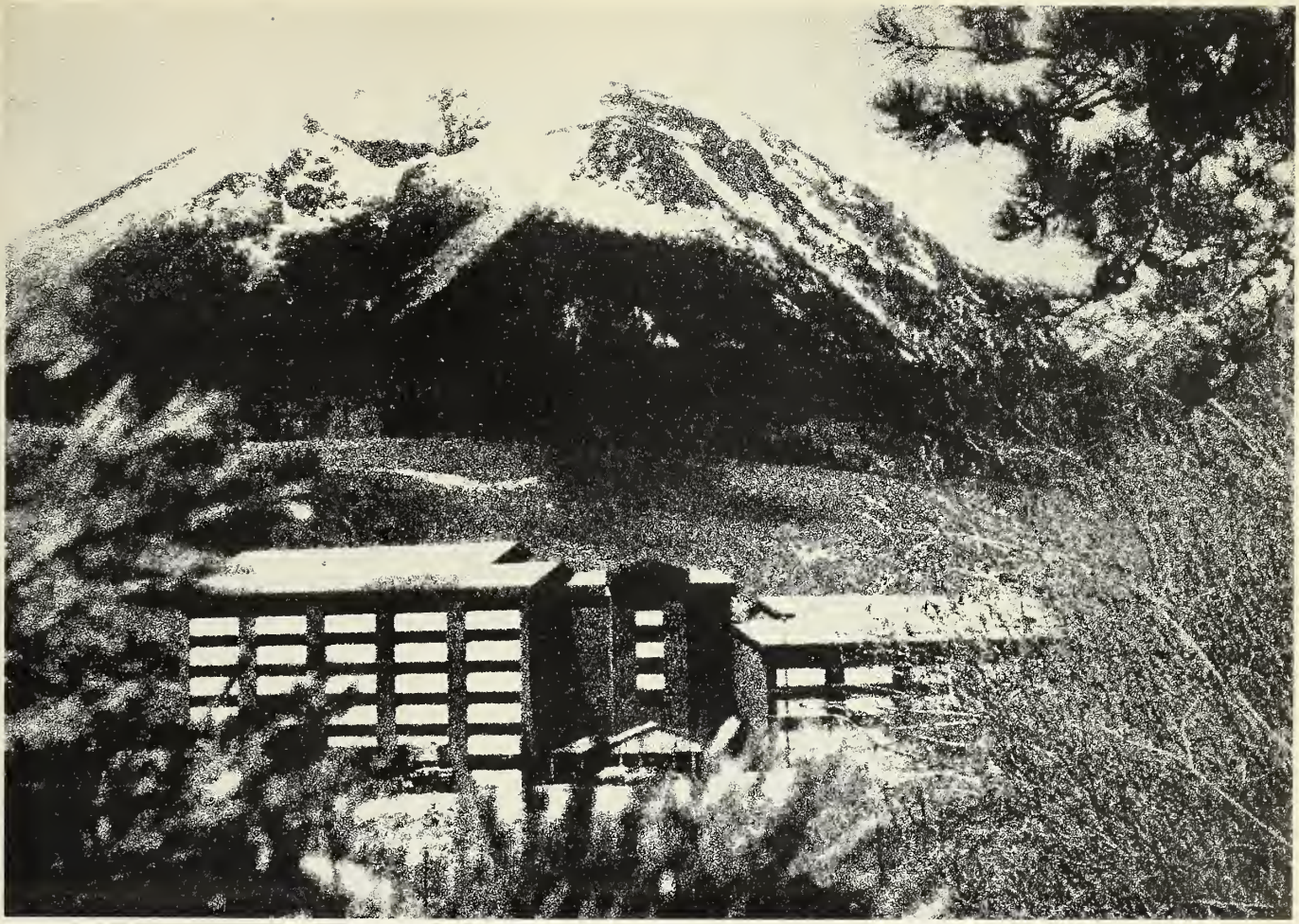
It was also announced that the Attorney General has subpoenaed from The Kansas Medical Society, Sedgwick County Medical Society, and the Kansas Hospital Association records relating to chiropractic; this relates only to an investigation at this time.

The Council endorsed Richard L. Brownrigg, M.D., for a second term on the Statewide Health Coordinating Council and directed that a letter to that effect be written to the Governor. It was suggested that a committee be appointed to monitor the Statewide Health Coordinating Council and to provide input to the Public Health Services Subcommittee.

The Council approved a contribution of $10,000 to MEDISERVE for 1981.

There being no further business, the meeting adjourned at 3:20 PM.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.



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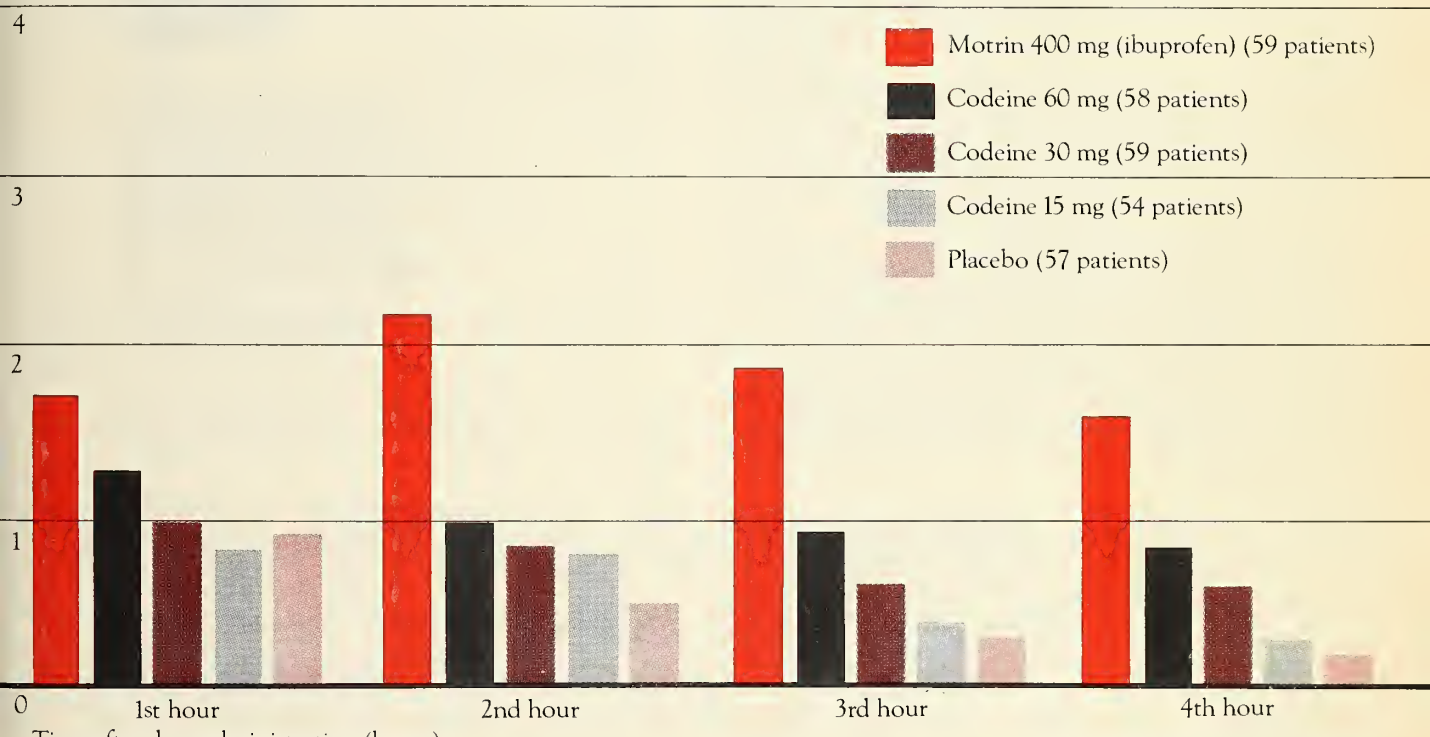
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Indications and Usage: Relief of mild to moderate pain.

Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

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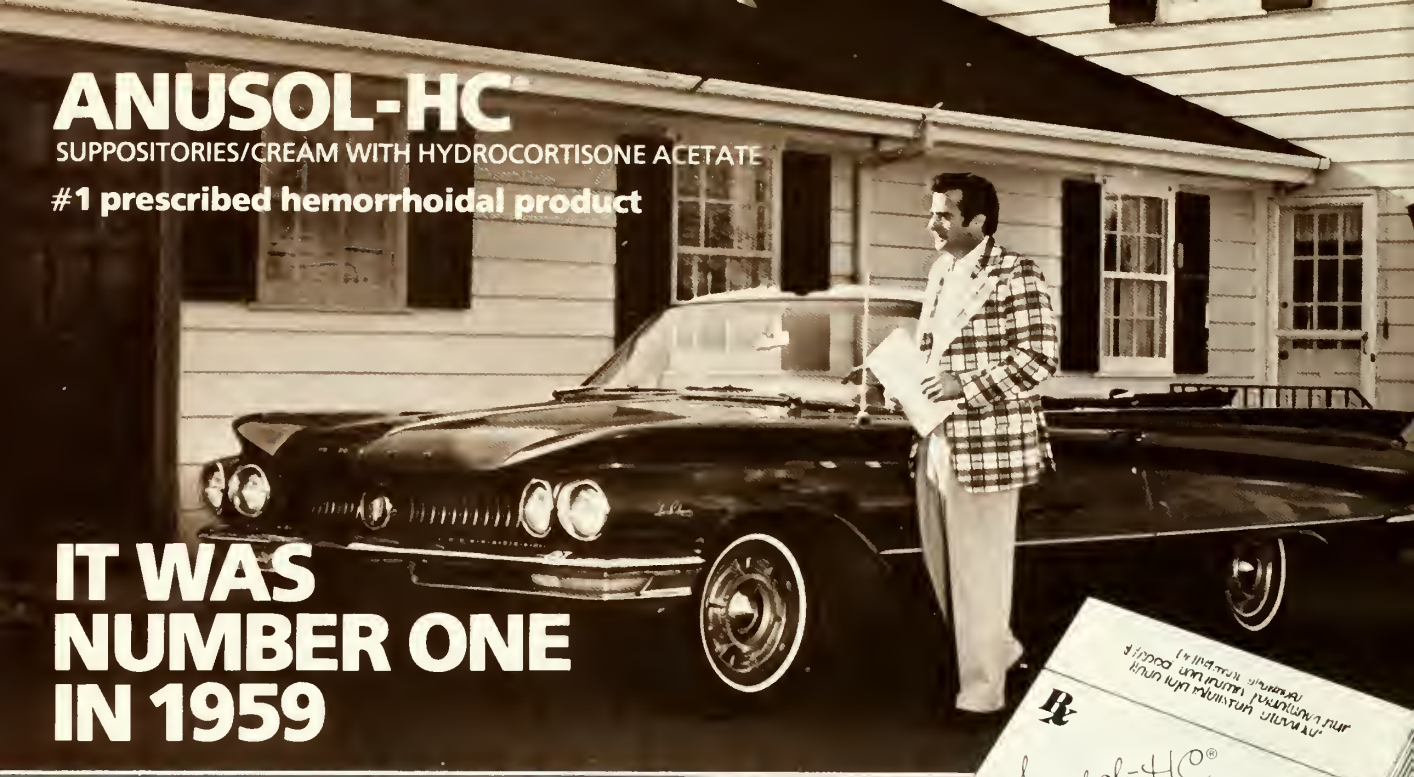
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Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE. If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

Store between 59°-86° F (15°-30° C).

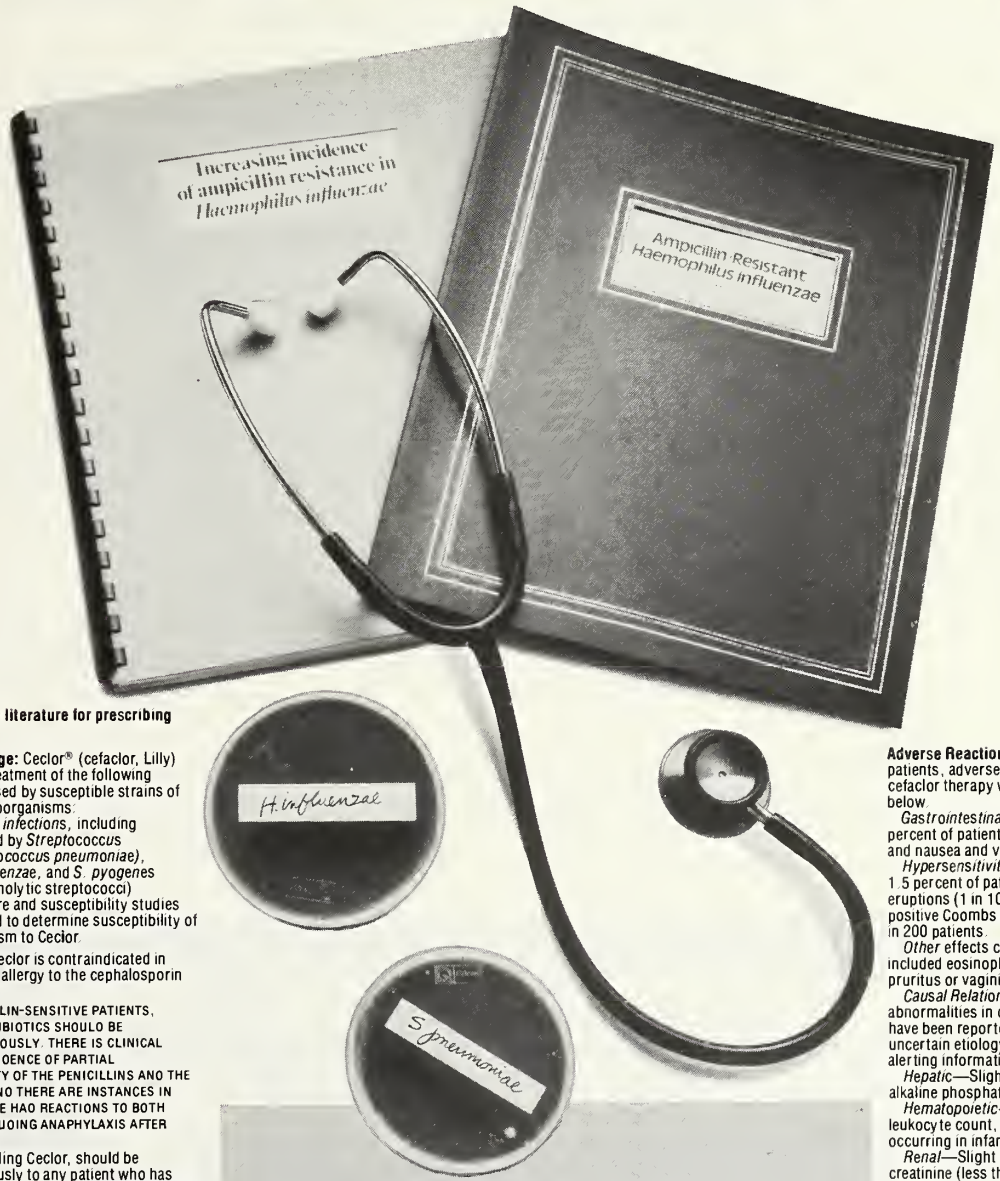
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An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor

Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions. If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about 2.5 percent of patients and included diarrhea (1 in 70) and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in about 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less than 1 in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [070379R]

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor® (cefaclor) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

1. Antimicrob. Agents Chemother., *8*:91, 1975.
2. Antimicrob. Agents Chemother., *11*:470, 1977.
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7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

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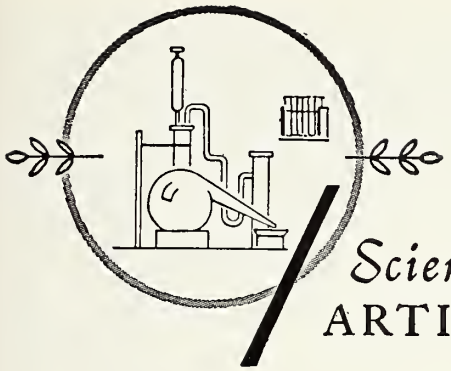
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Hemobilia Treated Radiographically

Angiographic Placement of Mini Coils

C. SOUCEK, M.D.; J. OXLER, M.D.; R. LAING, M.D.; T. ZINN, M.D.; W. BROOKS, M.D. *and* R. REEB, M.D., *Kansas City, Kansas*

Hemobilia from all causes, as reported by Sandblom in 1972, numbered only 545 cases, of which only 13 were reported as occurring subsequent to liver biopsy. These cases, as well as their treatment and results, are listed in *Table I*.[1-10] Since 1976, three cases have been treated by angiographic means with Gelfoam embolization[7-9] and one was treated with vasoconstrictors.[10] The case presented here is the first reported case to be treated with mini Gianturco coils. Large Gelfoam plugs (2 x 2 x 30 mm) were tried but were unsuccessful. The Gelfoam plugs simply passed through the arteriovenous shunt into the portal circulation.

Case History

A 37-year-old black male truck driver was admitted to Bethany Medical Center on December 5, 1979, for evaluation and treatment of multiple problems. He had been hospitalized 1½ years previously for severe anemia. At that time he had undergone extensive evaluation of the gastrointestinal tract; endoscopy, upper gastrointestinal examination, and barium enema revealed no abnormality. Bone marrow testing showed the anemia of a chronic disease. Liver-spleen scan was consistent with cirrhosis. He was lost to follow-up at that time.

From the Bethany Medical Center, Kansas City, Kansas.
Address reprint requests to Dr. Soucek, Radiology Dept., Bethany Medical Center, 51 N. 12th St., Kansas City, KS 66102.

On this particular admission, the patient entered with chest discomfort and palpitations. He was found to have a mild cardiomyopathy characterized by cardiomegaly and episodic atrial and ventricular arrhythmis. He had nephrotic syndrome with marked proteinuria and pedal edema, and serum albumin was 1.9. The nephrotic syndrome was thought to result from previous glomerulonephritis; however, no renal biopsy performed. He had hepatomegaly and a mildly elevated alkaline phosphatase of 44. SGOT, SGPT, and bilirubin were normal. Because of the possibility of multi-system disease such as amyloidosis, a rectal biopsy was performed and the results were normal. To further evaluate the patient's hepatomegaly and abnormal liver function, a liver biopsy was performed on December 17. This showed cirrhosis.

Hemobilia following liver biopsy is quite rare. In the case presented, angiography demonstrated a fistulous connection between the replaced right hepatic artery and the portal vein. This was angiographically embolized with mini Gianturco coils, producing cessation of the bleeding as well as a dramatic improvement in the patient's condition.

Two days following the liver biopsy, the patient developed right upper quadrant pain with radiation

TABLE I
TABULATION OF REPORTED CASES INCLUDING TREATMENT

Case	*Age*	*Sex*	*Delay to Onset (Days)*	*Treatment*	*Outcome*
1[1]	27	F	6	Mattress sutures	Good
2[2]	42	M	5	Biliary tract decompression	Alive
3[3]	26	M	2	Surgery; post necrotic cirrhosis	Alive
4[4]	40	M	2	Arterial ligation, T-tube decompression	Good
5[4]	59	M	2	T-tube Lap.; right hemihepatectomy	Died
6[4]	56	F	2	None	Good
7[5]	26	M	2+	None	Good
8[6]	16	M	6 & 30	Lap. T-tube Cholangiogram; surgically ligated left hepatic artery	Died
9[7]	43	F	7	Angio-Gelfoam	Well
10[8]	36	F	1	Angio-Gelfoam	Good
11[9]	16	M	10	Angio-Gelfoam	Good
12[10]	62	M	4	Epinephrine & Propranolol	Well
13[10]	33	F	10	Transfusions & Pitressin	Died
—	37	M	2	Angio-mini Gianturco coils	Good

into the shoulder and right lower quadrant. He had several episodes of melena over the next 24 hour period. Endoscopy performed the following day revealed esophageal varices, but they were not actively bleeding. No other abnormalities were present in the esophagus or the stomach. The duodenum was filled with bright red blood, but no mucosal lesion was identified. An arteriogram was performed but no bleeding site was identified.

During the next several days, the patient was treated conservatively with multiple transfusions of packed red blood cells, approximately two units/day. On December 23, duodenoscopy was repeated and, at this time, the patient was given Cholecystokinin. At this juncture, the blood was seen to spurt from the ampulla of Vater, and the diagnosis of hemobilia was established. During the following 48 hours, the patient's condition deteriorated dramatically with continued melena requiring numerous transfusions. He developed hepatic encephalopathy with asterixis. Liver function deteriorated dramatically. Bilirubin climbed to 5; SGOT to 60; SGPT to 68; and serum ammonia level to 192. On December 28, a repeat selective angiogram of the gastroduodenal artery revealed reflux into a replaced right hepatic artery with a hepatic artery portal fistula. Surgical consultation was obtained, and the surgeon felt that the patient would not survive hepatic artery ligation. The following day, after having received a total of 24 units of blood, the patient was referred to radiology for Gelfoam embolization of the right hepatic artery.

The patient was in hepatic coma with active bleeding and was being transfused. The superior mesenteric artery was catheterized without difficulty and a Number 7 French catheter was advanced out into the replaced right hepatic artery to the level of the arteriovenous fistula. Several large (2 x 2 x 30 cm) Gelfoam pledgets were injected; however these could be seen passing through the fistula into the portal vein. No small Gelfoam pledgets were injected since they would only pass into the portal system and occlude it. Two mini Gianturco coils were required to effect complete occlusion of the fistula. An angiogram following placement of the coils revealed complete occlusion of the fistula *(Figures 1 and 2)*.

The following day the patient was awake with no evidence of active bleeding. Blood pressure was stable and the patient wanted to go home.

The patient was dismissed on January 5, 1980, one week after embolization. He has been seen in follow-ups and continues to do well. Although he has a chronic anemia, with hemoglobin of 10, stool guaiacs are negative.

Discussion

Liver biopsy is a relatively safe procedure with a low mortality rate and is a very useful procedure in the diagnosis of liver disease. The risk of fatal complication following liver biopsy using the one-second technique has been estimated as one death/6000 biopsies. Other complications include bile peritonitis and bleeding, which is usually in-



Figure 1A. Superior mesenteric artery injection showing fistulous connection between the replaced right hepatic artery and the portal vein.

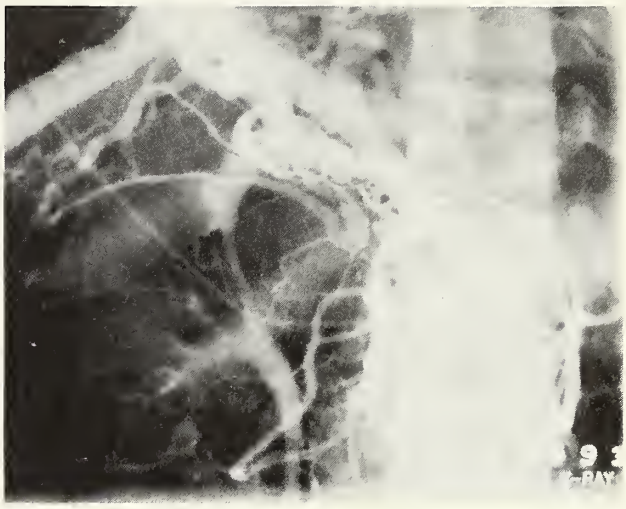


Figure 1B. Injection into gastroduodenal artery also shows reflux into the right hepatic artery with demonstration of the fistula.

traperiotoneal or subcapsular and is the leading cause of death. Hemobilia following liver biopsy is quite rare. The clinical diagnosis is suggested by the classical triad of biliary colic, gastrointestinal bleeding, and jaundice. There is frequently a two to seven day delay between the liver biopsy and the onset of hemobilia. Prompt diagnosis is important because of the serious nature of this complication which has a mortality rate of 40 per cent if untreated.

Endoscopic retrograde cholangiography or percutaneous transhepatic cholangiography may show filling defects in the bile ducts. Endoscopy is also helpful because it may show blood coming from the ampulla, thus confirming the diagnosis of hemobilia. Cholecystokinin may also be of use to stimulate biliary emptying. In this patient it definitely confirmed the diagnosis. Angiography, however, is the main diagnostic tool and it is very important to demonstrate the bleeding lesion. The angiogram may reveal an arteriovenous fistula as in this case or may demonstrate a fistulous connection between the hepatic artery and the bile ducts. Hepatic angiography may also demonstrate displacement of the branches of the cystic artery around a dilated gall bladder. The angiogram on this patient dehonstrated a dilated gall bladder.

From 1967 to 1976 the cases of biopsy induced hemobilia were treated by ligation of the hepatic artery drainage of the biliary ducts, or had no treatment. Since 1976 most cases have been treated with transcatheter Gelfoam embolization, which is much better tolerated by these debilitated patients than

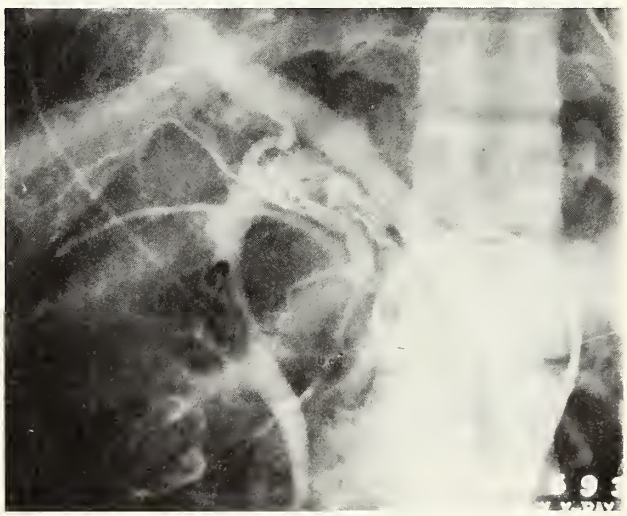


Figure 1C. Branches of the cystic artery are displaced by an enlarged gall bladder.

would be a surgical approach. The method is safe and directly occludes the responsible vessel. All cases treated by embolization have survived.

Hepatic artery ligation merely lowers the hepatic artery perfusion pressure. Due to extensive collaterals the blood supply to the liver is not completely occluded and bleeding may continue.

Transcatheter embolization has been widely used for various bleeding sites, and Gelfoam is one of the most popular embolic agents. In this patient the fistula was too large, and therefore this agent was unsuccessful.



Figure 2A. Selective injection of the replaced right hepatic artery.



Figure 2B. Catheter advanced to within 5 mm of arterial branch supplying the fistula.



Figure 2C. Two mini Gianturco coils inserted.



Figure 2D. Postembolization angiogram shows complete closure of fistula with increase in the collateral flow.

The safety of hepatic artery occlusion in a patient without cirrhosis is well documented in the literature. The great number of intrahepatic and extrahepatic anastomoses including capsular anastomoses is very important in preventing infarction. In the normal liver most of the blood supply comes from the portal circulation and so it is important to establish that the venous flow is intact with flow toward the liver. If there is marked portal hypertension with significant hepatofugal flow, embolization should be as peripheral as possible as the liver tissue supplied by the artery may undergo infarction. The use of short life embolic agents or the infusion of vasopressors could also be tried in an attempt to prevent infarction. Gelfoam is easy to use; however, it produces a relatively permanent occlusion.

Acknowledgement

St. Luke's Hospital and George D. Dixon, M.D., furnished the mini coils.

(Continued on page 306)

Resources

Diabetes Education in Kansas

RICHARD A. GUTHRIE, M.D.,* *Wichita, and*
JOSEPH L. KYNER, M.D.,† *Kansas City, Kansas*

DIABETES MELLITUS is a chronic medical problem. It may have its acute exacerbations which require professional treatment or even emergency, intensive hospital care, but for the long haul it is a chronic problem requiring daily maintenance and self-care. Much of the management is therefore dependent upon the patient, or the family in the very young. Accepting that one has this presently incurable medical problem for life, and then accepting responsibility for the care and learning about diabetes, can be very difficult for any patient.

An understanding, accepting patient makes the management task easier for the professional team. Crisis intervention and emergency admissions are less frequent, and patients and families tend to be happier and more satisfied when they understand and participate in their own care. They need to know what may be accomplished for themselves with good management.

Diabetes is common and increasing. Approximately 2-4 per cent of the population has the problem today, and it spares no age. Diabetic patients are a part of every physician's practice and they represent a significant percentage of hospital admissions. A basic, up-to-date understanding of diabetes and its management is therefore essential for the health professional. Most importantly, knowing who and what is available for specialized consultation or follow-up evaluation of the hard-to-manage or complicated diabetic will help to allay fears and unnecessary anxiety. As a minimum, patients with diabetes should learn where to seek information regarding their condition.

Personnel and Facilities

The Kansas Regional Diabetes Center has been formed recently in Wichita. While concerned with inpatient and outpatient care as well as clinical research, one of its important functions is education. A one week (5 day) educational program from 9:30 AM to 3:30 PM every other week is available to patients and their families. This educational program is in St. Francis Hospital in Wichita and is supervised by Richard Guthrie, M.D., of the Department of Pediatrics of the University of Kansas School of Medicine, and his staff. This group, including Husain Jawadi, M.D. (an internist), and two teaching nurses, Diana Guthrie and Debbie Hinnen, also participates in professional and public education endeavors in and outside of Wichita. The Center is supported by private funding through the University of Kansas Endowment Association. It accepts adults as well as children.

Diabetes mellitis is a common but often complicated medical problem. Help is available to the professional, the patient, and the public from a variety of sources; an overview of some facilities and institutions is presented and discussed.

The Cray Diabetes Education Center is a designated area in the new Bell Memorial Hospital of the University of Kansas College of Health Sciences in Kansas City. It is also supported by private funding through the University of Kansas Endowment Association. This Center provides the resources for outpatient and inpatient education. Together with the diabetes outpatient clinic, it is supervised by Joseph Kyner, M.D., of the Department of Medicine of the University of Kansas School of Medicine and his colleagues and staff. George Ann Eaks, R.N., is the primary teaching nurse. These, and other individuals in dietetics, pharmacy, podiatry and other fields, are all involved with various phases of patient and professional education, both in the hospital and throughout the state. This Center accepts primarily adults.

The Endocrinology Section of the Department of Pediatrics of the University of Kansas School of

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Medicine in Kansas City provides diabetes education to pediatric patients and others as necessary. Wayne Moore, M.D., and his staff in this department work closely with the Wichita Center and the Internal Medicine group in Kansas City to fulfill educational needs of the young diabetic patient.

Several community hospitals have designated areas for education and personnel to regularly perform diabetes teaching for their personnel and diabetic patients. This is an important development and is to be encouraged. Even a single nurse or dietitian in a clinic or office setting can greatly aid the physician by performing much of the patient education relating to chronic disease management.

Organizational Support

The University of Kansas plays several important roles in that the above centers are funded through its Endowment Association; its medical school and its hospital in Kansas City and affiliated hospitals in Wichita provide the resource centers; and its faculty and staff are available and active in diabetes education. The Division of Health Care Outreach of the University coordinates much of the state-wide education activities, particularly at the professional level. While the Division's Director (Joseph Meek, M.D.) is at the Medical Center in Kansas City, Health Education Centers have been established at Chanute (Southeast Region) and Hays (Northwest Region), and one is planned at Garden City (Southwest Region). These centers, while not having full time health professionals, can arrange and plan seminars, workshops, lectures, or teaching clinics utilizing faculty or staff from Kansas City or Wichita.

The American Diabetes Association performs an important advisory and coordinating role for the patient and the community. This volunteer health organization is active in Kansas with many chapters throughout the state. The Kansas Affiliate headquarters is in Wichita with Ellene Tompkins as Executive Director. The Kansas Affiliate is responsible for all counties in the state except Wyandotte and Johnson Counties, which are part of the Mid-America Affiliate of the American Diabetes Association. This latter group serves the greater Kansas City area and its executive director is Martha Lawrence.

Other community and volunteer organizations may regularly or at times support diabetes projects at one of the above listed centers. While not contributing directly to professional, patient, or community education, these volunteer efforts are vital for the necessary fund raising and financial assistance which in turn supports the educational endeavors.

Many professional societies and hospital administrations have facilitated the educational advances made thus far. Local and state wide groups can do much to implement programs and public awareness.

Professional Education

Programs for the practicing physician are provided in a number of formats ranging from the traditional circuit course program to physician participation in hospital or medical center teaching programs as an active observer. The latter type of educational experience can be individually designed for a week or longer at the Wichita Center or at the Medical Center in Kansas City. Physicians and nurses enroll in advance, and may choose an entirely didactic seminar type program, or a clinical experience.

Because of time and travel constraints on many practicing physicians and other professionals in their communities, the Regional Health Education Centers in Kansas are attempting to bring educational programs for the professionals to the local level. Such activities are initiated at the request of the physicians, nurses, dietitians, or hospital in that community. Because the Chanute Center was the first of these regional centers to become operational, more professional education by individuals or teams has been done to date in the Southeast Kansas area than elsewhere. However, the absence of a regional center in an area does not preclude physicians, members of their staff, or a team from visiting a professional group, hospital, or community anywhere in the state if invited. Some aspects of management and treatment can be completely appreciated only by first hand observation in a diabetes center or clinic, but the convenience of providing current information and review at the local level is recognized and implemented as extensively as possible.

Phone consultations are encouraged, not only between physicians but between nurses and other professionals regarding their diabetes problems. In the past, reaching a physician at the Medical School was sometimes difficult, and inaccessability was a common complaint of the practicing physician. Much of this has been alleviated by better trained secretaries and operators who facilitate calls to a professional, thereby securing an appropriate answer to the caller's question. The telephone continues to be an excellent educational device.

Patient Education

As noted, the Kansas Regional Diabetes Center in Wichita provides a week long educational program for patients and their families. Juvenile or adult patients may be referred by physicians or enter the program on their own initiative. Shorter term indi-

vidual and group education is also provided as needed for referred inpatients and outpatients in the Wichita area. The Cray Diabetes Education Center in Kansas City conducts a weekly self-management seminar in conjunction with the Internal Medicine Diabetes Outpatient Clinic at the University of Kansas Medical Center. Approximately 15 different subjects are covered in the educational series, and this is continually repeated. The public may attend these classes on their own initiative or by physician referral.

In many community hospitals and clinics, this type of instruction is being done increasingly by a designated nurse. Training and educating such an individual is of vital importance for effective and continuing patient education in diabetes. Patients and their families can gain tremendously from an intensive educational experience such as the one provided by the Kansas Regional Diabetes Center in Wichita. Financial, time, and distance constraints will not permit this program for every diabetes patient, however, and even for the patient who has had an ideal program of instruction and learning, continuous monitoring and reinforcement are essential. These are the aspects that a well trained and empathic professional at the local level can provide with regular classes and by being available for questions.

The American Diabetes Association, through its affiliates and chapters, has done much to emphasize education and provide diabetic patients and their families with resource materials and informational meetings featuring invited speakers. Fortunately, the Association has shifted its emphasis from detection (where there has always been much confusion and frustration) to research and education — more appropriate pursuits.

Diabetic summer camps are another American Diabetes Association project, which — when effectively managed — can provide a very important educational experience for the diabetic child. While the youngster with diabetes must learn that he or she is usually similar to a peer without diabetes in terms of capability, learning about diabetes and how it relates to them is the first step. The diabetic camp is an excellent place to learn while playing with others with similar diabetes problems. The Kansas Affiliate sponsors an annual camp at Rock Springs near Junction City. There is also a camp in the greater Kansas City area sponsored by the Mid-America Affiliate.

Public Education

Local press, television, and radio coverage of most of the educational events for professionals and patients noted above will advise the public or community that health problems (in this instance diabetes) are being adequately addressed. Items of national publicity or breakthrough announcements can alarm or cause unwarranted expectations, however, and for these reasons periodic, public information events are of value in the community. When incorporated with a day-long diabetes fair, for example (which is primarily for diabetic patients and their families), an open-to-the-public seminar or invited speaker can be very effective, particularly when questions from the floor are encouraged. Such fairs or seminars have been held recently in Hutchinson, Topeka, Kansas City and Wichita, and have generally been very successful.

A Common Cause or Team Approach

There is much that is still controversial and unanswered in diabetes. As with other medical problems, philosophies of management may differ. The means may therefore sometimes vary, but most importantly, the end point in terms of patient welfare remains the same. For this reason, effective diabetes education emphasizes the commonly accepted objectives.

Recognizing the priority of patient education as an important means of achieving effective diabetes management can result in cooperation at many levels. This is being satisfactorily accomplished in Kansas. There is still much to be done, but coordinating and unifying efforts among the University, the communities, the hospitals, the physicians and other professionals, and ultimately the patient, makes the task easier for everyone. The results of better management and a more satisfied diabetic patient population and public are well worth the effort.

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(Continued on page 300)

Thrombolytic Therapy

An Introduction and Review

DORAI THIAGARAJAN, M.D.,* ***Topeka***

Thrombolytic agents have only recently become available in the United States for treatment of thromboembolic disorders. Criteria for and effects of these agents are discussed, and various ramifications are reviewed.

In the treatment of thromboembolic disease conventional anticoagulants — *i.e.*, heparin or warfarin — while preventing further propagation of the blood clots, do not lyse the clots by themselves. Lysis of an intravascular clot may be more desirable than simple prevention of extension in certain clinical situations, *e.g.*, acute life threatening pulmonary embolism (PE). Thrombolytic agents — *viz* urokinase (UK) and streptokinase (SK) — that are capable of achieving this, although in clinical trial and usage elsewhere for nearly two decades, were only recently made available in the United States for the treatment of thromboembolic disorders. The mode of action, indication, dosage, and complications of these two agents are discussed briefly in this review.

Thrombolytic agents are essentially fibrinolytic agents, *i.e.*, capable of lysing fibrin (and fibrinogen), their action being mediated by conversion of an inert pro-enzyme called plasminogen to plasmin — the active fibrinolytic enzyme. When a clot forms intravascularly, a certain quantity of plasminogen gets trapped within the embolus or thrombus; SK and UK, by converting this trapped plasminogen to plasmin, eventually lyse the fibrin clot. However, these agents do convert free plasminogen circulating in the body to free plasmin, the latter being responsible for drug induced hemostatic defect secondary to hypofibrinogenemia, increase in fibrinogen fibrin degradation products (FDP), and destruction of certain other clotting factors such as factors V and VIII. Because of this potentially serious side effect, careful monitoring of certain laboratory coagulation parameters is generally considered mandatory during thromboembolytic therapy.

Streptokinase is derived from the filtrate of cultures of β-hemolytic streptococcus and is marketed as "Streptase" by Hoechst-Roussel Laboratories. Urokinase ("Abbokinase" — Abbott) is a polypeptide chain with a molecular weight of 54,000 currently manufactured by tissue culture technique. While UK converts plasminogen to plasmin directly, SK forms an intermediary complex with plasminogen which in turn converts the rest of available plasminogen to plasmin. It is of great importance to understand this basic difference in their mode of action as it is responsible primarily for several differences observed in clinical use between these two agents. Some of these are outlined in *Table I*.

The currently accepted indications for thrombolytic therapy include (1) acute *massive* PE with or without shock and (2) deep venous thrombosis (DVT), particularly of major veins such as ileofemoral and axillary veins. At the current state of knowledge, these drugs should not be used (abused) for trivial clinical situations such as superficial calf vein thrombosis. Again, the diagnosis of PE should be well documented and clinically life threatening.

Beneficial results have also been reported in other disorders such as A-V cannula occlusion and central

TABLE I
DIFFERENCES BETWEEN SK AND UK

	UK	*SK*
Plasminogen depletion	Rare (under 12 hrs of Rx)	Common
Plasmin excess	Rare (under 12 hrs of Rx)	Common
Dose adjustment	Simple	Titration required
Antigenicity	None	Yes
Cost	Very expensive	Relatively less expensive

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retinal vein thrombosis. Currently, available data do not support any conclusive clinical benefit of thrombolytic therapy in acute myocardial infarction. Miscellaneous clinical situations such as Budd-Chiari Syndrome and renal vein thrombosis have also been reported to be beneficially treated with these agents. Surgical embolectomy remains the treatment of choice for arterial emboli.

Both SK and UK are given intravenously. The usual loading dose of SK is 250,000 units given intravenously over several minutes (because of the possibility of immediate allergic reactions) followed by 100,000 units intravenously/hr, 24-48 hrs for PE and 72 hrs for DVT. Premedication with a parenteral corticosteroid such as 100 mg of Solu-Medrol intravenously may minimize the allergic and febrile episodes that often accompany SK use. It is advisable to assess the coagulation status of the patient by obtaining partial thromboplastin time, prothrombin time, platelet court, and thrombin time (TT) before beginning thrombolytic treatment. Thrombin time should be monitored periodically during the treatment course with SK and if it is maintained from two to five times the normal control value, no change in dose of SK is required. If TT is less than two times the control, the dose of SK should be decreased to 50 per cent and if TT is more than five times the control value, the dose of SK should be doubled. This latter situation indicates hyperplasminemia and by *increasing* the dosage, circulating free plasmin level is actually reduced due to the accelerated conversion of plasminogen and plasmin to the intermediary activator complex which binds the free plasmin. Ideally, the streptokinase resistance test should be performed *prior* to initiating therapy with SK as this will not only help in modification of dose of this drug, but SK itself may be contraindicated in an occasional patient who has a very high titer of antibody to this agent (presumably due to recent Streptococcal infection). In such a situation UK would be the drug of choice.

Urokinase is not antigenic and its dosage and titration are simpler. The usual loading dose is 4,400 units/kg intravenously followed by 4,400 units intravenously/kg/hr for 12-24 hrs. Therapy with UK is monitored by euglobulin lysis time which should be maintained between 15 and 20 minutes. Once UK or SK treatment is terminated, conventional anticoagulant therapy may be resumed, the dosage and time of initiation depending on the coagulation profile obtained after UK or SK is terminated.

The goal of thrombolytic therapy is to lyse the intravascular clots with minimal production of systemic fibrinolysis with the attendant problem of potentially serious hemorrhage. This can be avoided with the careful monitoring of coagulation parameters as alluded to previously. While other specific complications are negligible with the use of UK, allergic complications including anaphylactic reactions (2.4%), and febrile episodes in particular, frequently occur with SK therapy due to its antigenicity. Contraindications for thrombolytic therapy in general are the same as for conventional anticoagulants. In addition, these drugs are preferably avoided in patients with recent surgery or parturition (< 10 days), gastrointestinal hemorrhage (< 6 months), septic thrombophlebitis due to the danger of disseminating the infection and particularly in those with disordered hemostasis to begin with for obvious reasons. Arterial punctures are also discouraged while thrombolytic therapy is continued.

The availability of thrombolytic agents for clinical use has opened an entirely new mode of approach in the treatment of thromboembolic disorders. When compared to conventional anticoagulants, experience with these agents is certainly limited. Moreover, in view of their considerable expense and relatively increased frequency of hemorrhagic and other complications, the use of these agents should be restricted to approved indications. Careful patient selection also is important; in short, there is no justification for widespread abuse of these agents. Nevertheless, thrombolytic therapy may prove itself to be life saving and very valuable in the management of thromboembolic disease when used with caution and judgment. Further selected reading on thrombolytic therapy is available to interested readers.[1-6]

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Evaluation of Anemia

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Anemia, commonly encountered by the primary care physician, may not always be apparent on the basis of history and physical examination. The clinical problem may surface only following the return of screening laboratory data. The hematologist focuses on the blood smear in order to set a direction of laboratory investigation. With or without a blood smear the initial evaluation of anemia requires a logical, sequential approach in order to categorize the anemia. Tests can then be performed to discriminate which of the causes of anemia exist within a defined grouping. Haphazard laboratory investigation is neither productive nor cost-effective.

Who Is Anemic?

The 13, 12, 11 rule is probably a good one to follow; males with hemoglobin under 13 gms/100 ml, females under 12 gms/100 ml, and the pregnant patient under 11 gms/100 ml require further study. Most automated systems use 14 gms/100 ml for men and 12 gms/100 ml for women as the lower limits of normal. The probability of anemia in a nonpregnant female under 12 gms is approximately 80 per cent. Altitude, daily use of diuretics, as well as other factors, are mitigating circumstances to consider. Classification related to cause appears in *Table I;* clarifying examples (in parentheses) are included when indicated.

Reticulocyte Count

The first step in evaluating anemia is distinguishing the proliferative or nonproliferative group. The presence of young, newly-released red cells can be determined by the reticulocyte count. Methylene blue is applied to the red cells and those with an identifiable reticulum network are counted in reference to the other red cells present. The uncorrected reticulocyte count is reported as a percentage. Thus, 100 cells taking the stain of 1000 red cells counted would give a 10 per cent uncorrected reticulocyte count. Two additional manipulations of this percentage must be made. The first is the calculation of the *corrected reticulocyte count.* Since the reticulocyte counting refers to the number of red cells present, a decrease in the total number of red cells would falsely elevate the count. In the example being considered, if the patient suddenly dropped from 1000 to 500 red cells without changing the number of reticulocytes, the reticulocyte count would be 100 of 500 with a percentage report of 20. To obtain the corrected reticulocyte count, the patient's hematocrit divided by the normal hematocrit is multiplied by the uncorrected reticulocyte count.

As an individual becomes increasingly anemic, younger cells move from the bone marrow to the peripheral blood. The shift is proportional to the degree of anemia and related to the impact of erythropoietin on the bone marrow. The second correction is the *reticulocyte index* and requires familiarization with what is called the shift table. A hematocrit of 45 requires no correction; however, with a hematocrit of 35, the corrected reticulocyte count must be divided by 1.5; with a hematocrit of 25 by 2.0; and with a hematocrit of 15 by 2.5 as shown in *Table II.*

Armed with the reticulocyte index, the physician can place the patient in the proliferative group if the index is 3 per cent or higher and in the nonproliferative group if the reticulocyte index is less

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TABLE I
CLASSIFICATION BY CAUSE

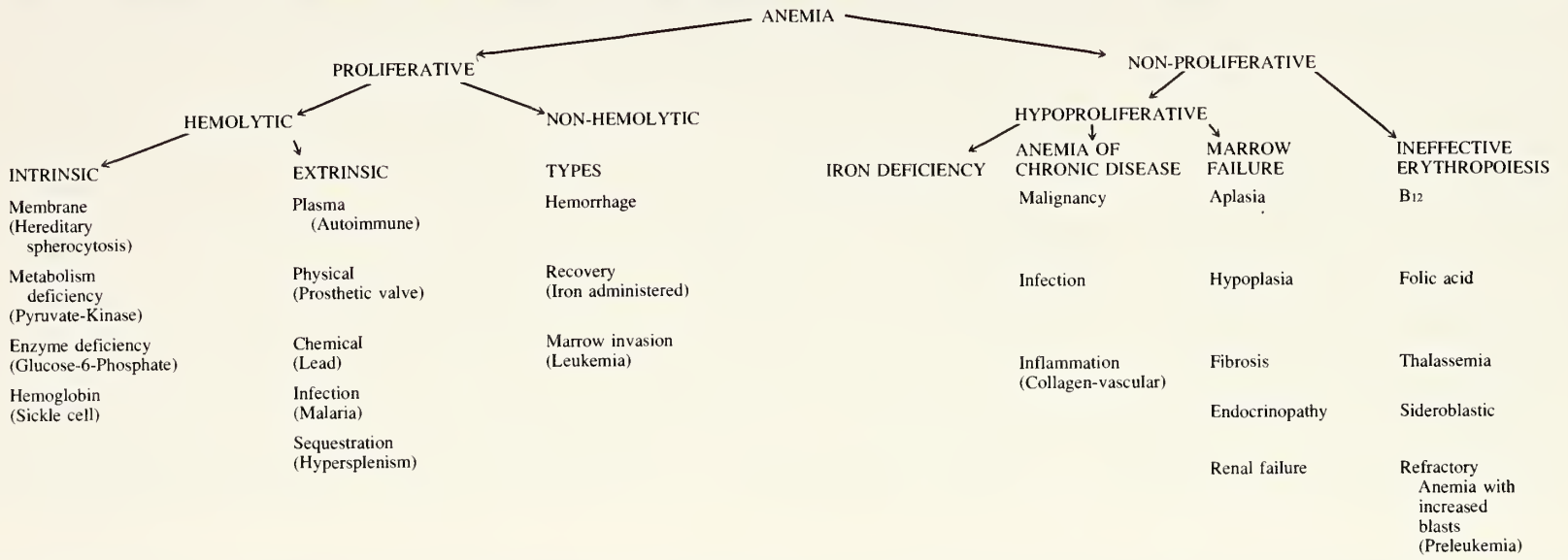


TABLE II
CORRECTED RETICULOCYTE COUNT

$$\frac{\text{Patient Hematocrit}}{\text{Normal Hematocrit}} \times \text{Reticulocyte Count} = \text{Corrected Reticulocyte Count}$$

RETICULOCYTE INDEX

$$\frac{\text{Corrected Reticulocyte Count}}{\text{Shift Table}} = \text{Reticulocyte Index}$$

than 3 per cent. Tests for serum B_{12} and folic acid need not be ordered for an untreated, stable patient with a reticulocyte index of 6 per cent because these deficiencies are examples of ineffective erythropoiesis of a non-proliferative anemia and the patient has a proliferative anemia.

Transferrin Saturation

To categorize further the causes for non-proliferative anemia, subdivision is obtained by utilizing the transferrin saturation. Serum iron is divided by the total iron binding capacity and the result is expressed as a percentage. As can be seen in *Table III*, patients with iron deficiency and the anemia of chronic disease yield test results of 20 per cent or less, whereas those with marrow failure or ineffective erythropoiesis have a transferrin saturation exceeding 20 per cent. The anemic patient with a transferrin saturation less than 20 per cent and a low serum iron would fall in the iron deficiency or anemia of chronic disease grouping. The total iron binding capacity (TIBC) may elevate in the iron deficient patient. When greater than 400 ng/dl there is excellent correlation with iron deficiency. Approximately 50 per cent of patients without stainable iron in the marrow will not have an elevated TIBC.[1]

Serum Ferritin

Anemia of chronic disease may be distinguished from iron deficiency non-invasively by measuring a serum ferritin. Serum ferritin, a circulating iron storage protein, correlates well with the amount of bone marrow iron. A low ferritin reading — less than 12 ng/dl — would be anticipated in a patient without stainable iron in the marrow. There are a few exceptions since it can be falsely elevated in patients with liver disease, ineffective erythropoiesis, and malignancy.[2] If the exceptions are considered, then a serum ferritin reading is a reliable, non-invasive way to distinguish between iron deficiency and the anemia of chronic disease.[3] Patients with malignancy, infection, or inflammation usually have increased iron in the marrow, normal or increased ferritin, and a normal or decreased TIBC. Marrow failure and ineffective erythropoiesis are characterized by an elevated serum iron and transferrin saturation.

Patients with proliferative disease almost always have a transferrin saturation greater than 20 per cent. The exception to this occurs when there is intravascular hemolysis. Iron is lost in the urine and can be recognized as urine hemosiderin. The serum iron, TIBC, ferritin, and marrow iron take on the characteristics similar to those of a patient with iron deficiency.

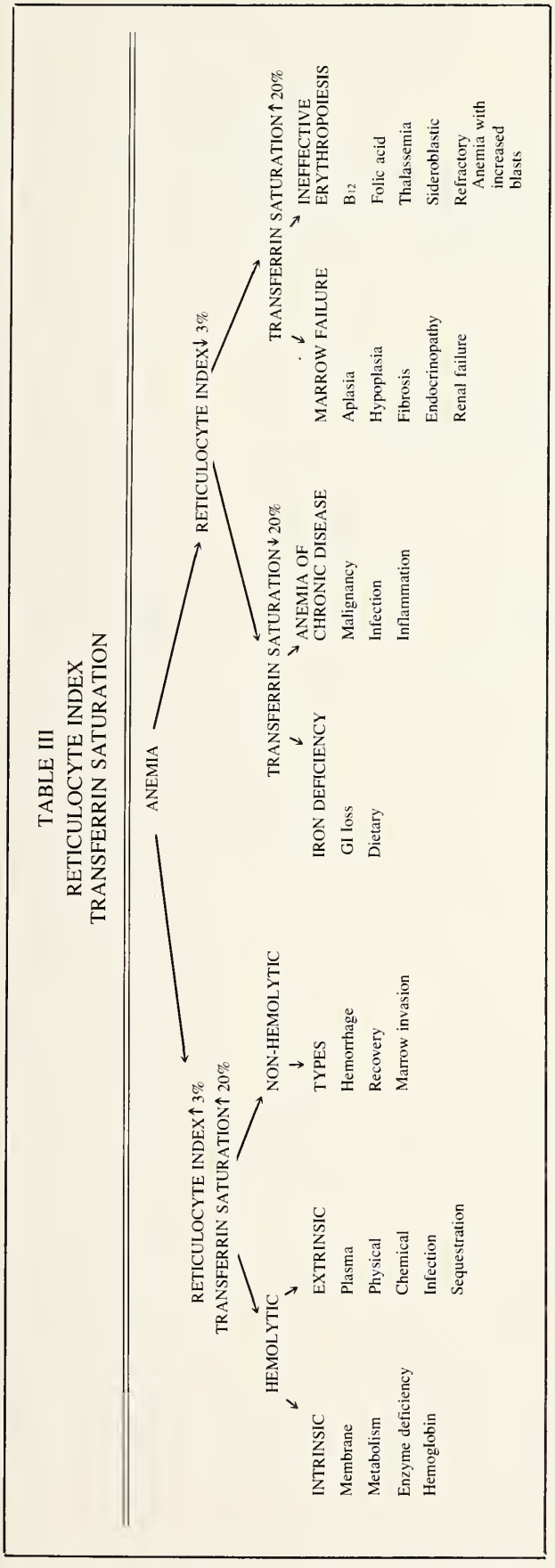
Red Cell Indices

When a blood count is requested, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) will normally be determined. The MCV is obtained by multiplying the hematocrit by ten and dividing it by the red blood cells expressed in millions. Automatic counting systems directly arrive at this number. If the patient has a hematocrit of 45 and red blood count of 5 million, then the MCV would be 45 times ten divided by 5.0, or 90. Since the MCH parallels the changes in the MCV, it is of little clinical value. The MCHC is obtained by dividing the hemoglobin in gms/100 ml by the hematocrit divided by 100. A patient with a hemoglobin of 15 gms/100 ml and a hematocrit of 45 would have a MCHC of 33.

The MCV will be reduced with intravascular hemolysis, hemoglobin C and E, iron deficiency, anemia of chronic disease, hereditary sideroblastic disease, and thalassemia. A decreased MCV, a mild anemia, and normal or slightly elevated red cell count point toward heterozygous beta thalassemia. The MCV will be elevated in extravascular hemolysis, non-hemolytic proliferative disease (in both cases, because of the appearance of younger, larger cells), B_{12} and folic acid deficiency, and often in acquired sideroblastic disease. The MCV is usually normal with marrow failure due to aplasia, hypoplasia, fibrosis, endocrinopathy, or untreated, uncomplicated renal disease. A decreased MCHC is seen in iron deficiency, anemia of chronic disease, hereditary sideroblastic disease, and thalassemia. The MCHC is elevated in only one circumstance, the presence of a significant number of spherocytes. *Table IV* illustrates in detail the changes described.

Special tests may be required to confirm a diagnosis or to elucidate causes of anemia. *Table V* details the various special tests that are performed for each category. A red cell survival is not listed as a test to be done for proliferative hemolytic disease because there are very few clinical indications for this test. Usually a patient with hemolysis has an elevation of the unconjugated bilirubin and the lactic dehydrogenase (LDH). The bilirubin level should decrease as the patient becomes anemic. As there are fewer red cells to be presented to the reticuloendothelial system for processing, the bilirubin should decline proportionately. A hemoglobin of approximately one-half of normal should be accompanied by a conjugated bilirubin of approximately one-half of normal. A high normal bilirubin in terms of the normal range and a profound anemia actually reflect an elevated bilirubin. In patients with hemolytic in-

TABLE III
RETICULOCYTE INDEX
TRANSFERRIN SATURATION



trinsic defects, enzyme analysis can be used for metabolic or enzyme deficiencies. Hemoglobin electrophoresis is indicated if an abnormal hemoglobin is suspected. There is little indication for osmotic fragility, although it is listed, because the fragile cells are the spherocytes and the non-fragile cells are those that contain little hemoglobin. These observations can be made on the blood smear without an osmotic fragility in most circumstances. For extrinsic hemolytic defects, Coombs' antibody and autoantibodies warm and cold can be utilized. The sugar water test is used for paroxysmal nocturnal hemoglobinuria, and an additionally useful screening test for this condition is the urine hemosiderin. Often patients with paroxysmal nocturnal hemoglobinuria will have urinary hemosiderin as evidence of intravascular hemolysis.

Special tests to distinguish intravascular hemolysis from extravascular hemolysis are plasma hemoglobin, urine hemoglobin, urine hemosiderin, decreased haptoglobin, and increased methemalbumin level. There are no special hematologic tests for the proliferative non-hemolytic group.

Special tests for the non-proliferative hypoproliferative group include determination of a decrease in bilirubin. Iron deficiency, of course, requires a careful hunt for the source of blood loss. Anemia of chronic disease does not have any special hematologic tests, but requires a careful clinical evaluation to identify the underlying disease process. Marrow failure, aplasia, and hypoplasia obviously require bone marrow confirmation, but in terms of special tests during the regenerative phase, both conditions are associated with an increase in hemoglobin F. Patients with bone marrow fibrosis often have an increased LDH. There are no special hematologic tests for the anemia of endocrinopathy or renal disease.

For non-proliferative ineffective erythropoiesis, the unconjugated bilirubin and LDH are both elevated as in the proliferative hemolytic group. In essence, patients with ineffective erythropoiesis can be thought of as having a massive destruction of the red cells in the marrow. Special tests for B_{12} deficiency include serum B_{12} and the Schilling test with and without intrinsic factor. Serum folate and red cell folate can also be determined. Special tests for thalassemia include an elevation of hemoglobin A_2 and hemoglobin F in beta thalassemia and decreased hemoglobin α to β chain synthesis in alpha thalassemia. Special tests for sideroblastic anemia require a search for the underlying causes. Diagnosis is confirmed on the marrow staining for iron demonstrating the ring sideroblasts so that no hematologic

TABLE IV

RED CELL INDICES/SERUM IRON AND TIBC/BONE MARROW IRON

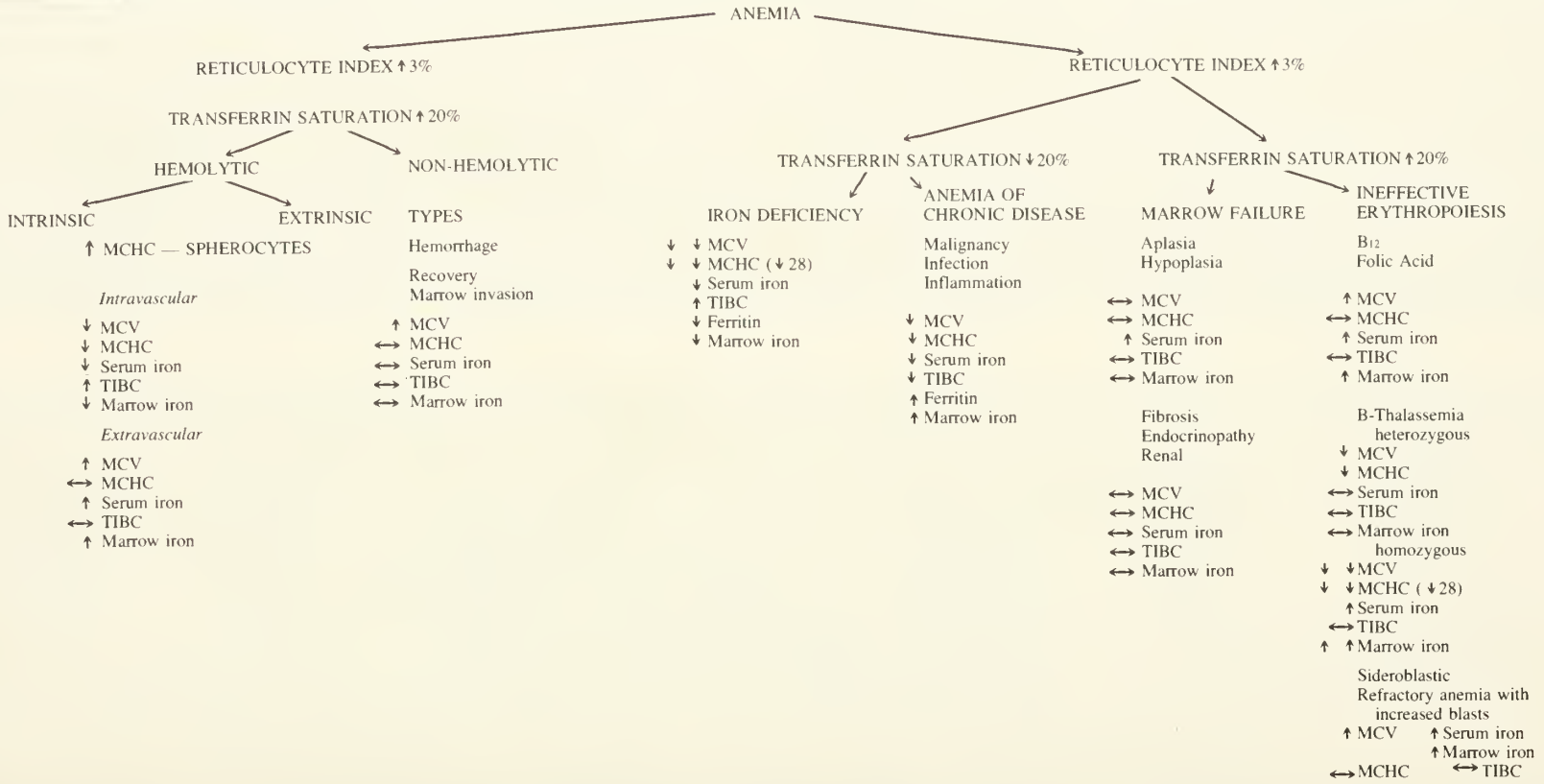
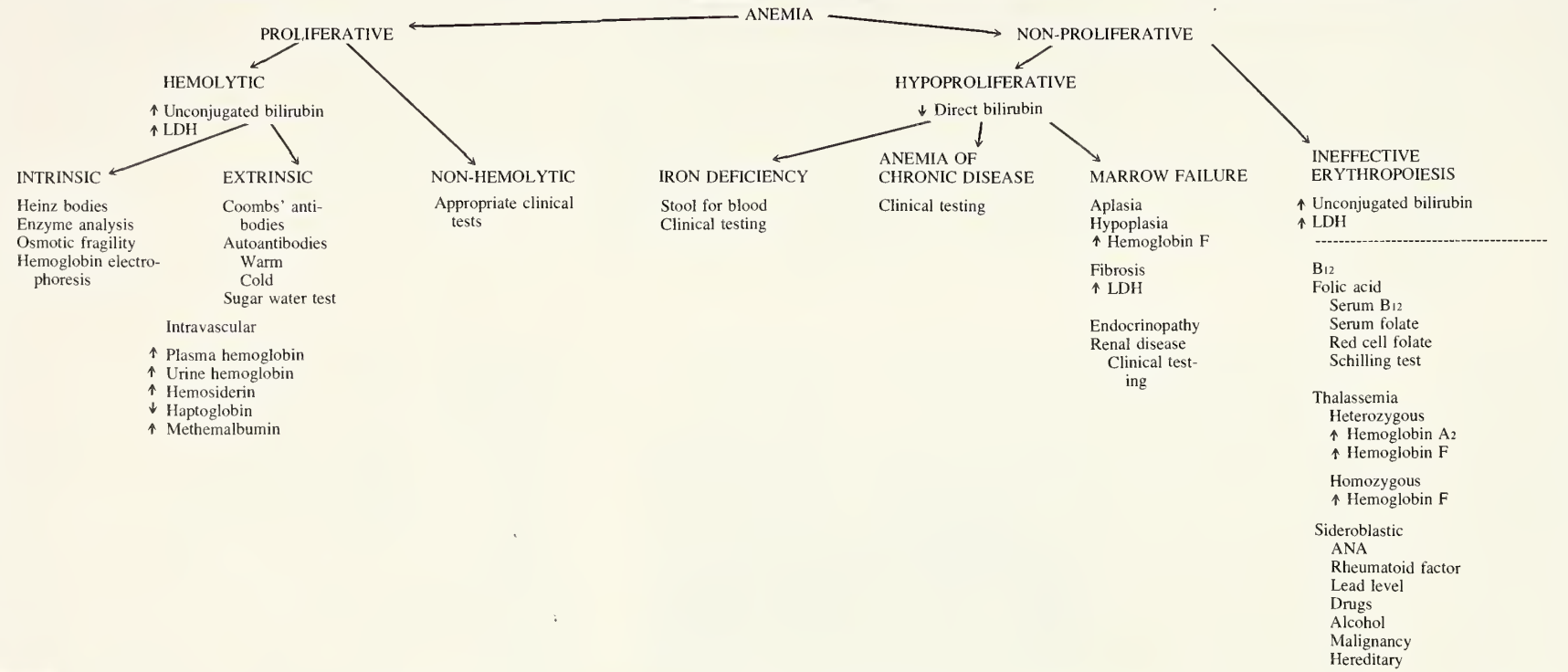


TABLE V
SPECIAL TESTS



tests beyond this are needed to elucidate the diagnosis. ANA, rheumatoid factor, blood lead level, historical search for drug ingestion and alcohol ingestion, search for malignancies and hereditary background may be clinically indicated.

Conclusion

About one-fourth of patients with anemia will experience acute bleeding, another one-fourth iron deficiency, and another one-fourth the anemia of chronic disease. All other causes of anemia make up the other 25 per cent with marrow damage, ineffective erythropoiesis, and hemolysis comprising the majority of this segment. These statistics reveal that 50 per cent of all patients will fall under the nonproliferative, hypoproliferative grouping. They will have a reticulocyte index of less than 3 per cent and a transferrin of less than 20 per cent. Anemia in the iron deficient patient can be repaired by therapy with obvious attention to the cause of the blood loss while the patient with anemia of chronic disease has ample iron stores. Anemia in the latter will improve only if the underlying condition can be eliminated or modified.

Students, residents, and private practitioners select levels of hemoglobin for the investigation of anemia that are too low 40-57 per cent of the time in women and 73-89 per cent in men.[4] Perhaps mild anemia is ignored because of its frequency. The application of a step-wise diagnostic approach may effect earlier evaluation.

Self-Assessment Questions

1. A patient with a hematocrit of 25 and corrected reticulocyte count of 4 per cent probably has a hemolytic anemia. (T or F)
2. A mean corpuscular hemoglobin concentration of 39 is diagnostic of spherocytosis. (T or F)
3. Non-hemolytic causes of a proliferative anemia are hemorrhage, recovery, and marrow infiltration. (T or F)
4. A serum iron test is helpful in distinguishing iron deficiency from the anemia of chronic disease. (T or F)
5. A reticulocyte index of 4 per cent excludes which of the following untreated conditions?
 a. Pernicious anemia
 b. Sickle cell disease
 c. Thalassemia
 d. Marrow aplasia
 e. Hereditary spherocytosis

(Answers on page 306)

Resources

(Continued from page 291)

Division of Health Care Outreach
University of Kansas Medical Center
Kansas City, Kansas 66103
Joseph C. Meek, M.D. (913) 588-4694

University of Kansas Health Education Center
P. O. Box 778
Chanute, Kansas 66720
Janet Perron, Director (316) 431-3050

University of Kansas Health Education Center
105 West 13th Street
Hays, Kansas 67601
Ruby Davis, Assistant Director (913) 628-6128

American Diabetes Association,
Kansas Affiliate, Inc.
2312 East Central
Wichita, Kansas 67214
Ellene Tompkins, Executive Director
(316) 265-6671

American Diabetes Association,
Heart of America Affiliate
616 E. 63rd, Suite 203
Kansas City, Missouri 64110
Martha Lawrence, Executive Director
(913) 361-3361

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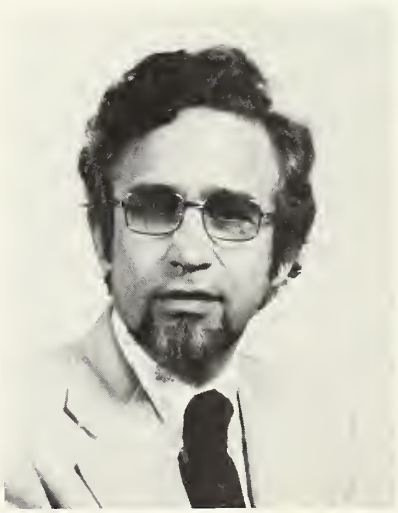
The President's Message

No one has to be told that we are living in the midst of an energy crisis. Fossil fuels are dwindling and the demand to find new sources of energy is increasing. If new sources are not found, disaster seems certain.

Fortunately alternate sources of energy for the Kansas Medical Society are more readily available. As those of us who are older (considered fossils by some) burn out, I am pleased to see new faces appearing on the scene at the House of Delegates. I assume this reflects new faces out in the counties, too.

In addition, I hope to tap two other sources which we have failed to fully utilize in the past. The first is an increased emphasis on recruitment and involvement of women physicians who represent 20 per cent or more of medical school classes these days. They are often among the brightest students and should make impressive leaders if we can give them the opportunity.

The other is the KMS Auxiliary. We all know how effective they have been in the AMAERF, health fairs, and similar projects. Their talents and education vary over a broad range of interests, and I believe that they can lend expertise to many of our committees. For this reason, with the cooperation of their president, Evelyn Huff, I am planning to incorporate them into as many committees as seems appropriate.



Times are changing. I hope you will join me in welcoming these women with open minds as well as open arms.

Fraternally,



President



Editorial
COMMENT

28.3495 Grams of Prevention

Someone had to go and discover Health.

Not that it hasn't been around for a long time — talked about and written about — but it has been one of those things like virtue — everyone knew about it and knew what they meant when they spoke of it, but define it? Well, it's — you know — like — umh. But now it seems we must come to grips with the matter of health — dissect it and analyze it, thereby (it is hoped) to define it in order to put it on a paying basis.

The pleasant but undefined condition operating up to this time has sufficed for less complicated times. Health was a condition one could drink to, wish someone good, brag or complain about, depending on the effect one wanted to have on one's audience, or, with religious remorse, report that there was none in us. We have taken it for granted when we had it and appreciated it, defined or not, only when we didn't.

This negative state of affairs is about to be changed and, as seems to happen not infrequently, the motivation to action stems not from the intrinsic worth of the condition but upon our need to translate it into monetary terms. Today, health, in addition to its other duties, must take on the task of cost containment.

It is not our intent to get involved in the complexities of medical care costs or the various efforts to reduce them. It is sufficient to note that the profession and public alike have been less than enchanted with the results of the various efforts to bring medical care costs down. True, the medical establishment has pointed with satisfaction when its figures have shown that voluntary efforts have shaved a fraction of a percentage point off the current general increase in prices. But these figures have at best indicated only that medical care costs are not going up as fast as some other things and haven't done much to comfort the patient. The frustration that has come from this chronic and resistant state has inspired the thought that if we can't significantly cut the cost of treating illness or injury after the fact, the answer is to re-examine our efforts to keep them from occurring and promote such efforts in a manner sufficiently attractive to interest the public.

Inevitably, this has focused attention on the apparent shortcomings of the medical profession in failing to do more to encourage health for its own sake. But physicians, preoccupied with the care of the sick and wounded, have assumed that their instructions in diet, living habits, and so on constituted an official and sufficient consideration of health as an entity. It is true that their health dicta have been therapy-oriented, and it hasn't, until the recent trend, been considered a dereliction of duty that health was not a recognized specialty complete with board certification and CME. It has seemed unnecessary to expound on the things taught in seventh grade Personal Hygiene I, to say once more that alcohol, tobacco, rich foods, late hours, and s-x could add up to trouble. It hasn't seemed likely the average patient would be happy to pay the price of an office call for such a recitation — even the annual physical examination has fallen on hard times in recent years. So, while limited efforts to make health appealing by lay groups or public health programs (loosely called "preventive medicine" in an earlier context) have gained some attention, by and large health has been the Edsel of medical practice.

But now, despite a seeming paradox in the demands, on the one hand, of unlimited freedom of choice of life-styles and, on the other, for medical regulations which will fend off the costly results, we find that the last great chance to cut medical care costs is for everyone to start paying attention to health. The effort is achieving status as it moves beyond the earlier concept of preventive medicine which, to the average physician, meant helping with the annual county immunization programs, fronting for the health agency fund drives, and generally pronouncing the virtues of health and the sins of unhealth (not necessarily supported by personal behavior). Various scenarios are being written for these efforts, and our admitted skepticism is bolstered by

the thought that it will be some time before any results can be evaluated. Complicated as the sociomedical scene is, there will be numerous claimants for the credit of any success in cutting medical care costs. Besides, no one seems to have considered that any appreciable reduction in medical care costs is going to have to be part of the resolution of our whole woeful economic picture since such costs are only part of that whole. If there is such success, everyone will be so happy over the general state of the economy they will be too busy indulging in unhealthy celebrations to give the medical contribution any credit.

Well, the idea of maintaining good health has merit in its own right apart from the economic impact. If good clean living can be made enticing and people can learn to choose parents whose DNA is straight arrow, we'll be on our way. In evaluating the effort, we can perhaps comfort ourselves that even if, as the critics say, the profession has been concerned only with curing disease, not preventing it, it has at least contributed to there being a lot more people around to be concerned about. The past successes of the medical profession in promoting the health effort have been absorbed into the social fabric, and the current criticism does seem to take on the tone of, "But what have you done for me today?"

Along the way, however, there are two related features that warrant our consideration. First, it would be well to keep in mind that, worthy as they may be in essence, many of the things included in this economic prophylaxis — nutrition is an excellent example — will be found laced through the cultist regimens where they are touted as things physicians don't know or don't appreciate or neglect in favor of their destructive routines. These points are exploited by our extraterritorial rivals as they apply to their distorted promotions their own brands of appealing but unsupported evidence which orthodox physicians, they sadly point out, don't understand. This approach has worked well for them through the ages, and a full-blown health promotion program, supported by the medical profession, the insurance companies, and the government, will give them added incentive (abetted by our current freedom-of-choice-even-if-it-kills-us climate). The potential of this may be appreciated by the recent pronouncement of one Michael Krisman, chief deputy director of California's consumers affairs agency which oversees the licensing of health professionals in the Golden State. Citing medicine's "professional imperialism," Krisman is quoted in *Medical World News* as saying, "The allopaths have managed to drive out naturopaths, homeopaths, and midwives in most states, and the allopaths have been trying to undercut osteopaths and chiropractors for years. We're into promoting all these different kinds of health care." The message should be clear: If we are committed to our patients' welfare as we claim, active involvement in the health promoting projects and an increasingly watchful eye on the cults will be essential.

The other aspect of the matter lies in the mechanics by which the medical profession can maintain a proper role in it. The purported lack of interest of the medical profession in health promotion in the past is a spurious charge since physicians have been more interested in promoting health than the public has been in having it promoted. Given this change of climate, if there really is one, the question of demand for health promotion against the needs for active medical care raises the problem of how a positive program can be implemented. There are, of course, numerous ancillary groups involved in the current health scene — nurse practitioners, physician's assistants, assistants in various therapeutic maneuvers. The relationship of these groups with physicians is at best uneven but two comments are consistent. Physicians say that they are overworked but at the same time the only ones equipped to handle the most demanding and technical responsibilities of their work. The ancillary groups say they do not want to intrude on or displace these responsibilities but fulfill responsibilities of their own. A well-developed, professionally supported, and valid program of health promotion would be an ideal place to utilize the training and experience of such individuals and still not deprive them of participation in active practice roles. Furthermore, such a system could make use of the particular rapport individuals in these areas of service are often able to establish with patients, a level of communication that patients often cannot establish with their physicians.

This type of preventive medicine is primarily a matter of education and dissemination of information derived from orthodox medical investigation and experience. What we are seeing is an attempt to use medical principles and service as an economic tool, an effort to make the human condition an economic asset before it becomes a medical matter in the traditional sense. Physicians cannot abdicate their responsibility in this area or choose only certain aspects of patient care they wish to acknowledge. A firmly bonded team of legitimate health professionals with the physician as the logical leader would seem to provide the appropriate agency for meeting this need and minimizing the intrusion of bureaucrats or other cultists. — *D.E.G.*

(Continued from page 273)



Roger Warren lays it on the line to the House of Delegates about Medicaid reimbursement.



Jesse Rising is rewarded for proving that twenty-seven years may not be forty, but it's longer than four!



Calvin Bigler makes a point about the relationship with ancillary groups.

—and the House of Delegates assumes an air of absorbed responsibility.





A portion of the Impaired Physician Program Committee exchange thoughts: Drs. Kermit Wedel, Erwin Janssen, Ivor Jones, and Max Teare in conference with Dr. and Mrs. Dick Draemel, Salina.

David Waxman, Executive Vice Chancellor, UKSM, accepts a check for AMAERF from the KMS Auxiliary passed on by President Don Goering—



—one way it came about: the AMAERF Auction, organized by Caryl Bichlmeier, stimulates both income and interest.



(Continued on page 318)

Hemobilia Treated Radiographically

(Continued from page 288)

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

Editor
The Journal of the Kansas Medical Society
1300 Topeka Avenue
Topeka, KS 66612

Evaluation of Anemia

(Continued from page 300)

Answers

1. *False*
2. *True*
3. *True*
4. *False*
5. *a, c, d*

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Official Proceedings

1980 Annual Meeting of the House of Delegates

Transactions of the 121st annual session of the Kansas Medical Society are published in this issue of *The Journal*.

The resolutions are printed in numerical order under the Minutes of the Second House of Delegates meeting. Those resolutions that were not adopted but were referred for further study for information are so indicated. The resolutions that failed to pass are retained in the official minutes at the Executive Office, but are not recorded here.

FIRST SESSION

The first session of the House of Delegates of the Kansas Medical Society met on Saturday, May 3, 1980, beginning at 9:00 AM, at the Glenwood Manor Motor Hotel, Overland Park, Kansas. The meeting was called to order by Clair C. Conard, M.D., Speaker, who introduced John W. Young, M.D., President of the Johnson County Medical Society, and John O. Yulich, M.D., President of the Wyandotte County Medical Society.

Doctors Young and Yulich welcomed the delegates to Overland Park on behalf of both the Johnson County and Wyandotte County Medical Societies, and extended an invitation to all delegates to attend the activities throughout the meeting.

Dr. Conard then introduced Mrs. Kathy Wedel, President of the Kansas Medical Auxiliary, who reported on Auxiliary activities during the past year, indicating that the area of membership had been addressed and that the theme of her year was the promotion of better health through education and healthful lifestyles. The Body Pollution Learning Center continue to enjoy increasing interest and a second center has been completed. Mental health has been added to the emphases of drugs, alcohol, tobacco, and nutrition. Requests for information regarding these programs have been received from groups in several other states who want to develop their own learning centers. The Auxiliary has developed a speaker's bureau, with Mrs. Peggy Null as advisor. This project will be coordinated with the efforts of the Kansas Medical Society. This past year the AMAERF raised a total of $17,714.54. The joint auction Friday evening raised a total of $4,616.75. The Auxiliary continued its legislative efforts with active participation in letter writing and contacting various legislators. Mrs. Wedel thanked the Kansas Medical Society for their continued support and for the opportunity to address this body.

The Honorable John Carlin, Governor, in addressing the House, stressed the many concerns in the area of health care and education, especially in view of today's economy. Resources are tight and there is limited flexibility. This necessitates the setting of priorities for the future. Governor Carlin indicated that while he may not always agree with the medical society, he welcomes input from Kansas physicians, whether it be concerning appointments or specific legislation. He complimented Jerry Slaughter on the excellent working relationship he has regarding appointments and legislation. He then thanked Dr. Goering and the House for the opportunity to visit this meeting and wished them a successful meeting.

Dr. Conard briefly reviewed the order of business and explained the composition of the House, indicating that it is composed of the elected officers, all past presidents, representatives of specialty societies, Councilors, and the duly elected delegates from component medical societies. He stated that the House would follow the *Sturgis Standard Code of Parliamentary Procedure*.

The Speaker then introduced the officers of the Society, as follows:

Donald D. Goering, M.D., Coldwater, President.

Phillip A. Godwin, M.D., Lawrence, President-Elect.

Herman W. Hiesterman, M.D., Quinter, First Vice President.

Kermit G. Wedel, M.D., Minneapolis, Second Vice President.

William K. Walker, M.D., Sedan, Treasurer.

Jack R. Cooper, M.D., Shawnee Mission, Constitutional Secretary.

Alex Scott, M.D., Junction City, AMA Delegate.

Clair C. Conard, M.D., Dodge City, AMA Delegate.

Lew W. Purinton, M.D., Wichita, AMA Alternate Delegate.

Kermit G. Wedel, M.D., Minneapolis, AMA Alternate Delegate.

Dr. Conard reported that an official quorum was present.

Upon a motion by Jack R. Cooper, M.D., Constitutional Secretary, the minutes of the 1979 annual meeting were approved by acclamation.

The following tellers were appointed for the primary election: Drs. Richard J. Cummings, Chairman, Wichita; Richard Meidinger, Topeka; and Charles S. Reeves, Fort Scott.

Dr. Purinton presented a plaque to Jesse D. Rising, M.D., Kansas City, in recognition of his many contributions in the area of continuing medical education.

Mr. Bud Wright, Director of Federation Communications of the American Medical Association, addressed the House, speaking on behalf of members of the Board of Trustees who were unable to attend. Mr. Wright reported that he works closely with the Kansas Medical Society staff, requesting input from the local level and providing information on various AMA activities. He stated that the AMA has scored some important victories in the courts and has strengthened its liaison with allied health organizations. Membership has increased by 12,000 during the previous year and dues will not be increased at this time. Growing attention is being addressed to the Washington scene, where efforts to increase control over the medical profession have continued. The Washington office has been reorganized and a new headquarters building is under construction. Congressional concern with health issues this year is anticipated to be at its lowest level in the past 15 years, because of the election year and the many inflationary factors. Mr. Wright spelled out four primary concerns in the area of legislation: (1) catastrophic health insurance, which is under continuing debate; (2) medical education and health manpower, in which the administration is recommending severe financial constraints on medical schools; (3) Medicare-Medicaid amendments; and (4) efforts to curb the Federal Trade Commission.

William K. Walker, M.D., Sedan, Treasurer, presented his report. Printed copies of the report were distributed to every member of the House. Dr. Walker stated that while expenses increased, it will not be necessary to raise dues for the coming year.

In concluding the Editor's Report, Dr. Gray presented Dr. Goering with a bound volume of *The Journal* for the President's term 1979/80. Dr. Goering in turn presented the Editor with a bound volume of *The Journal* for 1979.

Jimmie A. Gleason, M.D., Topeka, Chairman of the Legislative Committee, stated that because the Legislature is still in session at this time, he cannot present a final report. He thanked the committee for the work it had done this past year in monitoring a total of 77 bills. He then briefly reviewed the following bills:

HB 2750 — *Hospital Cost Containment*. This bill would have provided for hospital rate review by the Department of Social and Rehabilitation Services for those hospitals who did not participate in the Blue Cross Rate Review Program. The bill was killed this year, but it will probably be reintroduced in the next session.

SB 542 — *Physician Advertising*. The bill as proposed by the Advertising Committee under the chairmanship of Howard N. Ward, M.D., Topeka, was completely rewritten to cover 17 professional groups with the exception of lawyers. The bill as rewritten would provide that any advertising was acceptable as long as it was not false, fraudulent, misleading, or deceptive. This rewritten version was not acceptable to the Medical Society. It was being held in committee. However, HB 2705, dealing with circuit courts, was rewritten completely to include provisions of the advertising bill. This proposal also is being held in committee.

HB 2731 — *Open Records Act*. This is a proposal to open Medicaid records and make information available on physicians participating in the program and the monies they had been paid. A conference committee voted to remove this amendment. However, the Senate rejected the conference committee report. The bill will be tabled for this year.

HB 3252 would have deleted the "reasonable" clause from the Medicaid program. The bill is presently held in committee.

Dr. Gleason reported that next year there will be consideration of bills relating to confidentiality and cost containment by the state. He encouraged physicians to support KaMPAC and promote candidates for election.

Ronald B. Davis, M.D., Wichita, Chairman of KaMPAC, emphasized the need to increase the membership in KaMPAC in order to become more effective. He criticized the increasing government regulations and impersonalization of physician-patient relationships. He stated that KaMPAC can help stop this trend and help reverse increasing governmental encroachment into the practice of medicine. To be effective, however, KaMPAC needs physician involvement and support.

Herman W. Hiesterman, M.D., Quinter, reported on the activities of the KU Admissions Committee. He described the selection process and emphasized how fair, precise, and meticulous this process is. He commended the KU staff for their cooperation and assistance. Dr. Hiesterman stressed the importance of the personal interview in order to get to know the person on an individual basis. He praised the process



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Bronchial asthma	Sulfonamide hypersensitivity

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work/play—normal hemodynamic responses to exercise maintained.

love—low incidence of impotence and/or loss of libido: 2.8% in 1,923 patients studied.[1]

cardiac output—tends to return to control values during long-term therapy.

blood flow—preserved in kidney.

No Single Advantage Determines Drug Choice.

Other factors must include:

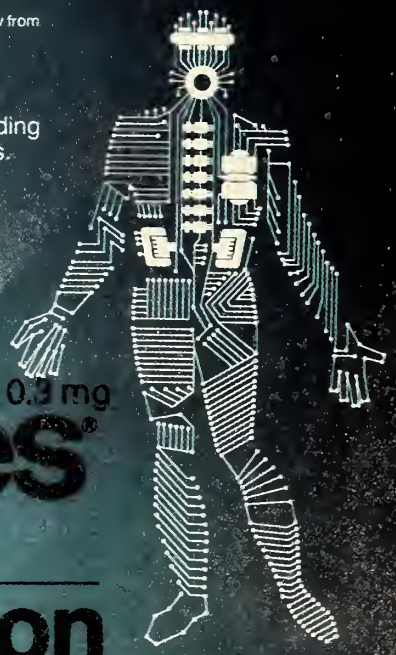
The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

1. Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

Now available in new 0.3 mg tablets

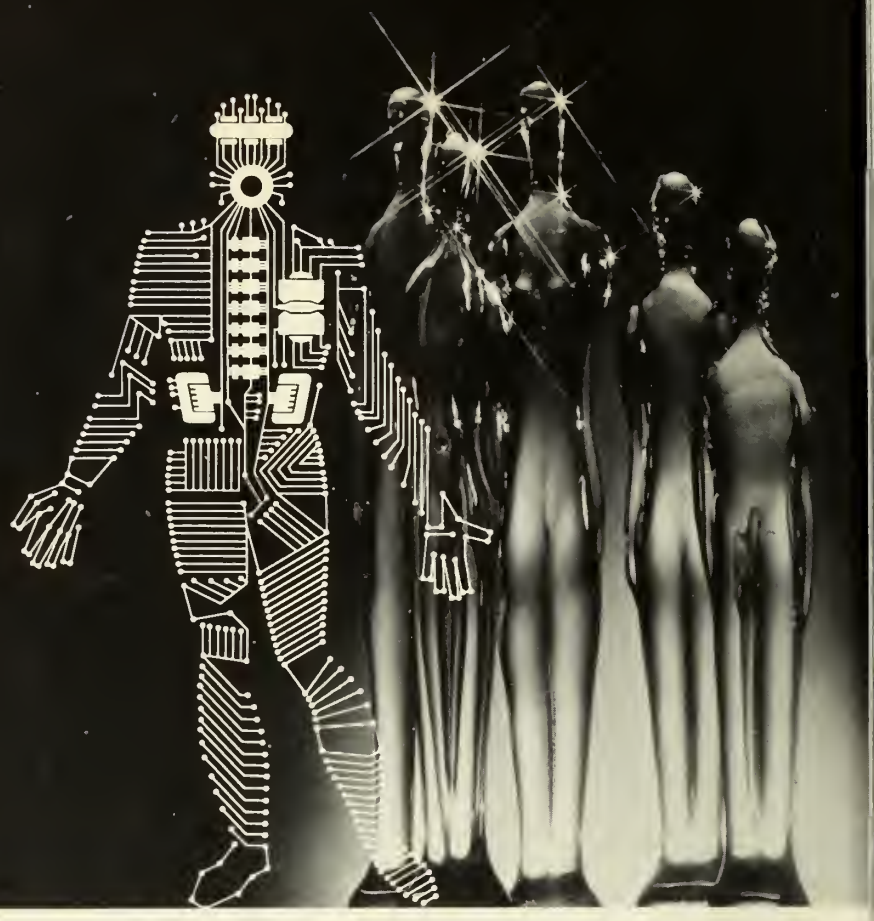


Tablets of 0.1, 0.2, 0.3 mg

Catapres®
(clonidine HCl)
Hypertension

The Alpha Advantage:

It's for all kinds of hypertensives



Tablets of 0.1, 0.2, 0.3 mg

Catapres®
(clonidine HCl)

Hypertension

- No contraindications.
- Effective in all degrees of hypertension. It is mild to moderate in potency.
- Low incidence of depression, impotence, orthostatic hypotension—no fatal hepatotoxicity.
- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
(clonidine hydrochloride)
Tablets of 0.1, 0.2, 0.3 mg

Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
Under license from Boehringer Ingelheim GmbH

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Boehringer Ingelheim Ltd.
Ridgefield, CT 06877

and encouraged physicians to participate in future committees.

K. William Bruner, Jr., M.D., Wichita, Chairman of the Nurse Practitioner Committee, reported that the 1978 Legislature passed a law providing for Advanced Registered Nurse Practitioners, giving complete authority to the State Board of Nursing to develop regulations. The Kansas Medical Society believes that the ARNP should function as a member of the physician-directed health care team and opposes independent practice of ARNPs without physician supervision. Previous drafts of the nursing regulations by the Board of Nursing went beyond the legislative intent and were rejected by the Legislature in January 1980. An amended version of the regulations has been drafted since that time. These regulations have been accepted by the Legislature and will go into effect as temporary regulations for a period of one year. While the Board of Nursing will be preparing the permanent regulations during this one-year period, the Kansas Medical Society must be prepared to monitor and provide input into the development of these permanent regulations.

Emerson D. Yoder, M.D., Denton, Chairman of Mediserve, Inc., reviewed the history and establishment of the Mediserve program. Kansas Medical Society and the Kansas Farm Bureau have each been contributing $10,000 per year to the program. Additionally, county farm bureaus and county medical societies have provided contributions. The primary purpose of the Mediserve program is to provide financial assistance to medical students in return for primary care services in rural Kansas. Mediserve program also provides admissions assistance through recommendations to the Admissions Committee. Prospective medical students are interviewed to determine whether they should be granted admissions assistance or financial support. When the program was originally established, Chancellor Archie Dykes promised 20 slots to Mediserve for each entering class. A total of 34 students have been admitted to the freshman and sophomore classes in the past two years. For 1980, a total of 12 Mediserve recommendations have been admitted with two alternates. Mediserve is currently working with Chancellor Dykes to improve the impact of Mediserve recommendations and the admission process.

Franklin G. Bichlmeier, M.D., Kansas City, Chairman, reported that the basic committee structure of the Judicial Committee has been completed and that legal counsel is presently drafting procedures to ensure due process. Several complaints have been addressed by the committee as of this date.

Colonel James W. Wilson, M.D., Fort Riley, reported that there is a severe shortage of physicians in the armed services and encouraged physician participation in the reserves or active service.

Theodore E. Young, M.D., Topeka, reported that the principal task of the Welfare Advisory Committee has been to advise Secretary Robert Harder concerning the Medicaid program. Dr. Young briefly reviewed the results of the Medicaid survey of Kansas physicians. The committee has attempted to encourage payment of reasonable fees and care of patients in the physician's office instead of referring them to treatment in a hospital emergency room. The committee has met with very little success. Physicians are continually frustrated with the program and are contemplating possible withdrawal from the Medicaid program, or considering action similar to that undertaken by the Nursing Home Association.

The president, Donald D. Goering, M.D., Coldwater, announced that Jerry Slaughter, Executive Director, was not present as he was still in Topeka monitoring the Legislature before its last hours of adjournment. He extended Mr. Slaughter's apologies for not being present at this meeting.

Dr. Goering reported that the Professional Liability Commission was successful in amending the Health Care Stabilization Fund Act that would require new physicians to pay 25 per cent surcharge the first time they participate in the Fund. The Commission is also looking into a study of risk management for Kansas.

Dr. Goering reported that it has been an excellent year for him. He thanked the KMS staff, the various committees, and the membership for the support and work they have done this year. He asked for continued support for Dr. Godwin in the coming year.

Dr. Goering stated that there are presently no physicians in the state legislature. This being an election year, he encouraged physician participation and support of candidates who are friends of medicine.

Primary election results for the office of second vice president were as follows: Jimmie A. Gleason, M.D., Topeka, and Lew W. Purinton, M.D., Wichita.

Resolution No. 80-19, Physician Hospital Relationships, was placed on the Consent Calendar.

The Speaker announced the following appointments to the Reference Committee: Drs. G. Rex Stone, Chairman, Manhattan; J. Roderick Bradley, Greensburg; William W. Burney, Wichita; Dean T. Collins, Topeka; and John W. Young, Shawnee Mission.

The House heard the following reports, after which the session was adjourned at 11:50 AM.

Necrology Committee — David E. Gray, M.D., Chairman

At this point we pause to reflect on our colleagues who have died since our last meeting. In epitaph, I offer the following words from Emily Dickinson's poem, "Death":

Because I could not stop for Death,
He kindly stopped for me.
The carriage held but just ourselves
And Immortality.

Name and Address	*Age*	*Date*
Rene A. Alonso, *Topeka*	58	Oct. 11, 1979
John H. Basham, *Eureka*	67	Apr. 23, 1979
Harold Bowman, *Wichita*	75	Oct. 6, 1979
Richard H. Claiborne III, *Baxter Springs*	51	June 16, 1979
Richard F. Conard, *Emporia*	63	Apr. 8, 1980
George E. Cowles, *Wichita*	83	Dec. 7, 1979
Murray C. Eddy, *Hays*	81	Apr. 30, 1979
Alfred S. Hawkey, *Newton*	75	May 16, 1979
Charles E. Henneberger, *Atwood*	93	Aug. 28, 1979
John D. Huff, *Kansas City*	57	Sept. 16, 1979
Norman E. Hull, *Hays*	55	Jan. 3, 1980
Joseph H. Johnson, *El Dorado*	80	Sept. 27, 1979
Henry W. Kassel, *Kansas City*	73	Feb. 28, 1979
Irene Koeneke, *Halstead*	81	July 12, 1979
Eugene J. McCreight, *Liberal*	79	Oct. 10, 1979
W. L. Pratt, *Leavenworth*	74	Apr. 27, 1979
Robert G. Rate, *Halstead*	65	Oct. 25, 1979
A. S. Reece, *Gardner*	78	Apr. 8, 1980
Albert J. Rettenmaier, *Kansas City*	84	Nov. 26, 1979
Edward J. Ryan, *Emporia*	67	Dec. 14, 1979
Glen R. Shepherd, *Kansas City*	59	Sept. 1, 1979
Galen M. Tice, *Kansas City*	80	Sept. 22, 1979
Eugene A. Walsh, *Onaga*	61	Nov. 5, 1979
Gene V. Williams, *El Dorado*	55	July 4, 1979

Editorial Board — David E. Gray, M.D., Chairman

Again, it is time for the annual accounting in which your sundry committees explain what they have been doing all year by which you are supposed to decide whether they should do it again next year. Although the Editorial Board is beholden to the Council for its survival, it recognizes its need for all the friends it can get, so we approach you with our usual shining countenances and winsome ways, hoping that you have found favor with our efforts.

Since there is nothing like a comfortable financial position to promote good will, we are pleased to call attention to the financial report which is still printed in black. Inflation hasn't been repealed, the Post Office keeps trying to make a profit, and our publisher maintains his becoming attitude of remorse over the increasing cost of paper and ink. The result is that our bank balance is showing the same erosive effect of the east coast as it is battered by relentless tides but has not yet started to imitate the mud slides of the west coast.

We do bring to your attention one matter which will be causing us come collective head scratching during the coming months. You are well aware of the activity being generated in the area of physician advertising by the unseemly attitude of the Federal Trade Commission. This will be giving your Editorial Board something of an ideologic workout and, while hardly on a par with the Middle East situation, it poses some problems whose solutions, we anticipate, will warrant the advice of the membership.

My personal satisfaction in making these reports lies in the opportunity it gives me to remind you that the Board is indebted to some very nice people for the fact that you get your journals at all. Eleanor Bell is approaching veteran status as her good judgment and function become an increasing asset to the *Journal.* As for the Managing Editor, there is really not much to say. Outside of promoting and developing and arranging the substance of the *Journal,* overseeing its production, nursing its finances, keeping the Editorial Board in line and working, coordinating the work of numerous ancillary groups of the Society, and saving the Editor untold time and embarrassment and criticism, she mostly sits around. The Business Manager maintains his tolerant vigil on our business affairs including countersigning the checks to discourage the Editor from such modest embezzlements as might be possible. He continues to fulfill a father-figure role to the Board — since any member of said board is old enough to be his father.

It has long been my purpose at this time to extend the good wishes of the Board to this body in its deliberations, prompted by the sobering thought that the parliamentary progress of our Society is a direct function of the durability of your posteriors. To sustain you, I offer the advice of a wise lady of the last century. At the time the House of Lords was considering the question of Irish home rule, Lord Ribblesdale was scheduled to confront Lord Salisbury in a crucial debate. On the night before the debate, Lady Ribblesdale is reported to have sent her husband the following telegram: "Hit him hard, hit him often — and always below the belt."

And now, in keeping with our heart-warming tradition, the Editorial Board is pleased to present a bound copy of the year's journals to our president, Don Goering, whose "President's Page" has pro-

vided our readers with information, inspiration, and a warning that they were coming to the Editorial Comment.

The Kansas Foundation for Medical Care, Inc. — Louis M. Culp, M.D., President

At the present time, the Kansas Foundation for Medical Care has delegated PSRO review to 133 of the 145 acute care hospitals in Kansas. PSRO review would be implemented in all 145 Kansas hospitals were it not for a "Halt further PSRO implementation order" which was issued last September by the Health Standards and Quality Bureau of the Department of Health, Education and Welfare.

The order stated, in essence, that because of funding limitations at the national level, no further implementation may take place by PSROs until further notice. It is estimated that further implementation will not take place until the 1981 federal fiscal year which begins October 1, 1980.

I would like to emphasize that the halt order was, in no way, a reflection on the operation of the KFMC. The KFMC had scheduled to attain full implementation by November 1, 1979; and further, the KFMC's financial position is sound. The halt order, rather, was a reflection of an unfavorable total national funding level in place when the order was issued.

Another national issue affecting PSROs is the continuing debate surrounding the issue of confidentiality of PSRO data. As you are probably aware, the United States Tenth District Court in Washington, D.C., ruled that the National Capitol Medical Foundation, the PSRO for the District of Columbia, must release certain provider identifiable profiles of practice patterns under the Freedom of Information Act.

This ruling is being appealed, and affects only the PSRO in Washington. It is estimated that the appellate process on this issue will take two to five years. In the interim, I should point out that there are several bills pending in congress which, if enacted, would exempt PSROs from the release of information under the Freedom of Information Act. Additionally, the United States Supreme Court acted on March 4, 1980, on an issue similar to the PSRO confidentiality issue. In that ruling, the court stated that organizations funded by federal grant (such as PSROs) are not agencies of the federal government, and therefore are not bound to release data under the Freedom of Information Act. We feel that the March 4 ruling expresses the intent of the court in a clear manner, and we look for a favorable resolution of the confidentiality issue within the next year.

It is well to point out that the KFMC does not maintain information that is identifiable to any particular physician. Rather, the information the KFMC compiles and maintains is identifiable only by a unique identifying number assigned to each physician by the hospital. Only the hospital knows which number is assigned to each physician.

Since I spoke to the House of Delegates last May, the KFMC met all of its stated grant objectives, except for those objectives which we were precluded from meeting by the enactment of the "Halt implementation" order last September.

The KFMC's 1980-81 grant year objectives, as approved by the KFMC Board of Directors, were sent out in March to the chief of the medical staff of each Kansas hospital, as well as the chief executive officer of each Kansas Hospital. The board and staff would welcome any comments you may have regarding these objectives.

The KFMC presently has a staff of 22. In September, Alex C. Mitchell, M.D., Lawrence, joined the staff as medical director.

With the growth in the number of staff, it became necessary to move the executive offices to new office space in January. The board and staff would like to invite you to visit the offices when you are in Topeka. The new location is at 1000 West 10th Street.

At its March meeting, the board authorized the staff to initiate activities in the area of private review. Private review would be that review conducted for non-federal third-party payors such as health insurance companies. We believe that by becoming actively involved in the area of private review, the KFMC can obtain some fiscal flexibility as well as broaden our review base.

Another recent development is in the area of modified review. On April 1, 1980, the first phase of the KFMC's modified review plan went into effect in delegated hospitals. The greatest benefit from this activity lies in possible elimination of review where appropriate.

As of May 1, 1980, KFMC membership has grown to 1,850 members. This represents an increase of 30 per cent in one year. If you are not a member, and would like to join, please contact the KFMC office.

The board of directors and the physician committees of the KFMC continue to be active. Eight new board members were elected this morning, and the following officers were re-elected:

Treasurer — George R. Learned, M.D., Lawrence

Secretary — L. William Halling, M.D., Hays

Vice President — Richard M. Glover, M.D., Newton

President — Louis M. Culp, M.D., Kansas City

The KFMC can continue its successful operation only through the participation of Kansas physicians. The officers and Board of Directors invite your active participation in KFMC activities.

SECOND SESSION

The second session of the House of Delegates of the Kansas Medical Society was called to order by the Speaker, Clair C. Conard, M.D., at 9:00 AM on Sunday, May 4, 1980, at the Glenwood Manor Motor Hotel, Overland Park.

The Speaker placed some rules before the body and announced that the *Sturgis Code of Parliamentary Procedure* would be followed at this meeting. Every delegate would be given an opportunity to be heard on every question but except for the person who makes the motion, all delegates were asked to cooperate by being heard only once upon a single question.

The presence of a quorum was announced.

The following election results were reported:

PRESIDENT-ELECT: Herman W. Hiesterman, M.D., Quinter

FIRST VICE PRESIDENT: Kermit G. Wedel, M.D., Minneapolis

SECOND VICE PRESIDENT: Jimmie A. Gleason, M.D., Topeka

CONSTITUTIONAL SECRETARY: Jack R. Cooper, M.D., Shawnee Mission

TREASURER: William K. Walker, M.D., Sedan

AMA DELEGATE 1981/82: Alex Scott, M.D., Junction City

AMA ALTERNATE DELEGATE 1981/82: Kermit G. Wedel, M.D., Minneapolis

SPEAKER: Clair C. Conard, M.D., Dodge City

VICE SPEAKER: John O. Yulich, M.D., Kansas City, Kansas

The newly installed president, Phillip A. Godwin, in his address to the House, reported that some concern had been expressed about the number of family practitioners serving on the Executive Committee. He pointed out that he is board certified in Anesthesiology and also board certified in Family Practice. He practices in a group of three family practitioners and employs a nurse clinician. Dr. Godwin assured the House that the Executive Committee represents a broader perspective than is commonly thought.

In briefly reviewing his plans for the coming year, Dr. Godwin indicated that he plans to coordinate efforts in addressing ARNPs, Physician's Assistants, midwives, pharmacists, physical therapists and other groups, so that the medical society has a coordinated position. A letter has been sent to all members inviting them to serve on a committee of their interest within the Kansas Medical Society. A good response to that letter has already been received.

Dr. Godwin announced council district elections as follows: District 6 — James H. Ransom, M.D., Topeka; District 7 — Marvin D. Snowbarger, M.D., Emporia; District 10 — Herbert Fransen, M.D., Newton; Richard A. Siemens, M.D., Lyons, Alternate; District 12 — Philip J. Antrim, M.D., Anthony; Daniel J. Suiter, M.D., Pratt, Alternate; District 16 — John Neuenschwander, M.D., Hoxie; John Rand Neuenschwander, M.D., Hoxie, Alternate; District 18 — Robert W. Hughes, M.D., Lawrence.

Dr. Godwin then installed Clair C. Conard, M.D., as Speaker and John O. Yulich, M.D., as Vice Speaker of the House of Delegates.

Dr. Conard thanked Dr. Stone and the Reference Committee members for their efforts in summarizing all opinions on the resolutions.

There being no further business, he adjourned the House at 12:45 PM.

An asterisk following the resolution number indicates a change in the Constitution and By-Laws.

RESOLUTION NO. 80-1
Physician's Assistants — Supervision

Resolved, That it shall be the Kansas Medical Society policy that the personal presence by a supervising physician in the Physician's Assistant's activities where the services are rendered should be the standard of care in the usual practice setting; and be it further

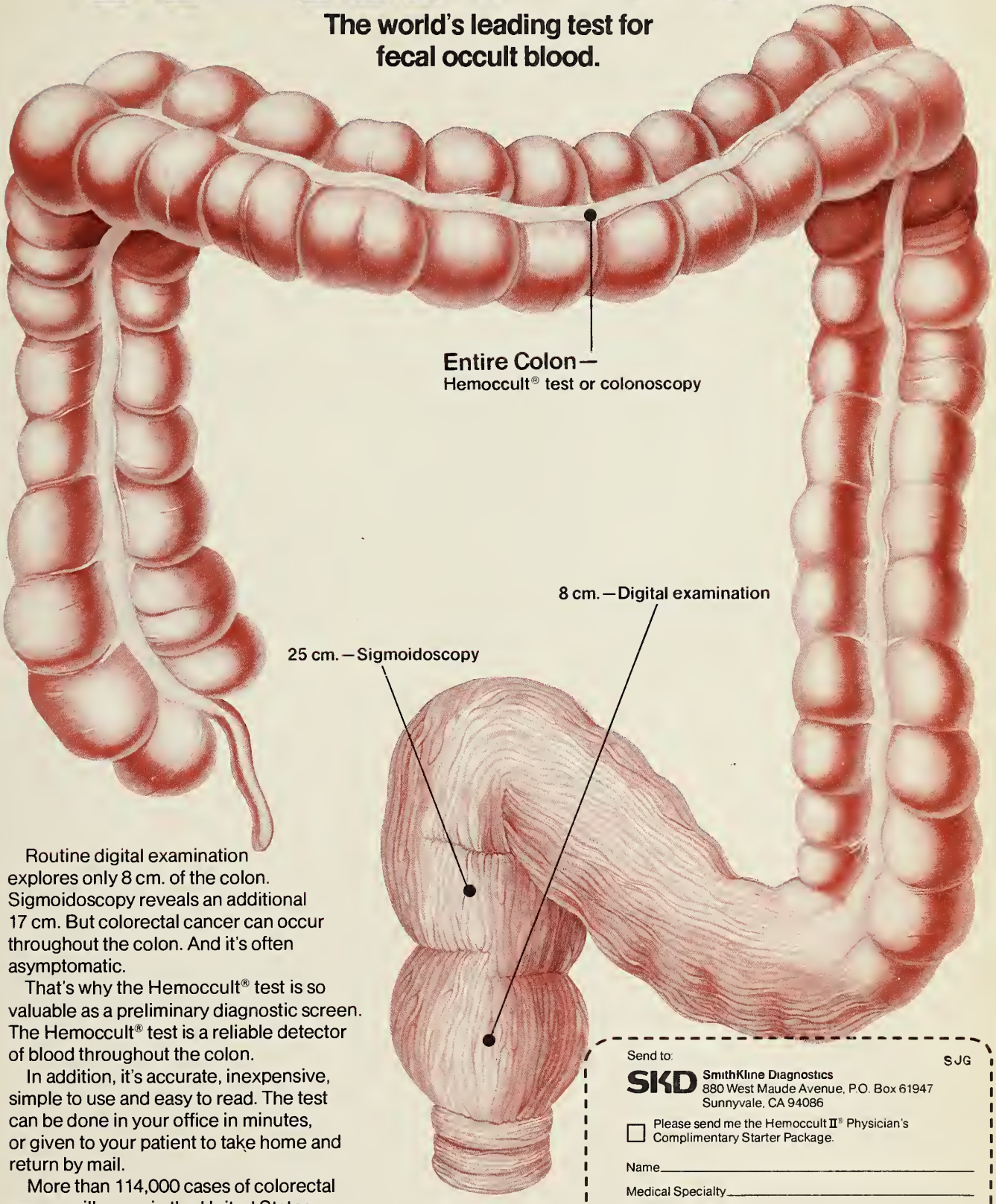
Resolved, That in unusual practice settings, exceptions to the standard of care should be determined and a specific waiver to this standard may be granted by the State Board of Healing Arts on an individual case-by-case basis, after annual approval by the local county medical society.

RESOLUTION NO. 80-2
Scientific Basis of Medical Practice

Resolved, That the Kansas Medical Society holds that the diagnosis and treatment of diseases such as essential hypertension, heart disease, stroke, cancer, diabetes, and infections by practitioners attributing

Hemoccult®

The world's leading test for fecal occult blood.



Routine digital examination explores only 8 cm. of the colon. Sigmoidoscopy reveals an additional 17 cm. But colorectal cancer can occur throughout the colon. And it's often asymptomatic.

That's why the Hemoccult® test is so valuable as a preliminary diagnostic screen. The Hemoccult® test is a reliable detector of blood throughout the colon.

In addition, it's accurate, inexpensive, simple to use and easy to read. The test can be done in your office in minutes, or given to your patient to take home and return by mail.

More than 114,000 cases of colorectal cancer will occur in the United States this year. The earlier they are diagnosed, the greater the chances for successful treatment. Send for your free Hemoccult® starter package, today.

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Perdiem™

empties the bowel gently...

by filling it.

Perdiem™ . . . the re-educative laxative . . . relieves constipation by a unique combination of physiological bulk stimulus and gentle pharmacologic encouragement of peristaltic response.



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(Please see next page for prescribing information)

Perdiem™

Prescribing Information

ACTIONS Perdiem™ with its gentle action, does not produce disagreeable side effects. The vegetable mucilages of Perdiem™ soften the stool and provide pain-free evacuation of the bowel. Perdiem™ is effective as an aid to elimination for the hemorrhoid or fissure patient prior to and following surgery.

COMPOSITION Natural vegetable derivatives. A unique blend of psyllium and senna (Plantago Hydrocollaid with Cassia Pod Concentrate).

INDICATION For relief of constipation.

PATIENT WARNING Should not be used in the presence of undiagnosed abdominal pain. Frequent or prolonged use without the direction of a physician is not recommended. Such use may lead to laxative dependence.

DIRECTIONS FOR USE—ADULTS Before breakfast and after the evening meal, one to two rounded teaspoonfuls of Perdiem™ granules should be placed in the mouth and swallowed with a full glass of warm or cold beverage. Perdiem™ granules should not be chewed. After Perdiem™ takes effect (usually after 24 hours, but possibly not before 36-48 hours), reduce the morning and evening doses to one rounded teaspoonful. Subsequent doses should be adjusted after adequate laxation is obtained.

IN OBSTINATE CASES: Perdiem™ may be taken more frequently, up to two rounded teaspoonfuls every six hours.

FOR PATIENTS HABITUATED TO STRONG PURGATIVES: Two rounded teaspoonfuls of Perdiem™ in the morning and evening may be required along with half the usual dose of the purgative being used. The purgative should be discontinued as soon as possible and the dosage of Perdiem™ granules reduced when and if bowel tone shows lessened laxative dependence.

FOR COLOSTOMY PATIENTS: To ensure formed stools, give one to two rounded teaspoonfuls of Perdiem™ in the evening with warm liquid.

DURING PREGNANCY: Give one to two rounded teaspoonfuls each evening.

FOR CLINICAL REGULATION: For patients confined to bed, for those of inactive habits, and in the presence of cardiovascular disease where straining must be avoided, one rounded teaspoonful of Perdiem™ taken once or twice daily will provide regular bowel habits. Take with a full glass of water or beverage.

FOR CHILDREN: From age 7—11 years, give one rounded teaspoonful one to two times daily. From age 12 and older, give adult dosage.

NOTE: It is extremely important that Perdiem™ should be taken with a plentiful supply of liquid.

HOW SUPPLIED: Granules, 100 gram (3.5 oz.) and 250 gram (8.8 oz.) canisters.

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all such ailments to misalignment of the spinal vertebrae is a hazard to health and that a patient who relies on such practitioners in the treatment of such ailments may delay proper medical care and serious and irreversible damage may occur.

RESOLUTION NO. 80-3*

Official Policy of the Kansas Medical Society

Resolved, That official policy on a given subject established through resolutions at the House of Delegates shall be the most recent action taken on that subject. All other prior resolutions dealing with that subject shall be rescinded and deleted as per Resolution No. 66-43; and be it further

Resolved, That the bylaws be amended with the addition of Section 5.444

Expiration

Official policies established through resolutions at the House of Delegates shall be in effect for a period of five (5) years at which time that policy position will be reviewed by the Executive Committee and will expire subject to approval by the House of Delegates unless superseded or continued by another resolution.

RESOLUTION NO. 80-4

Continuing Medical Education Accreditation

WHEREAS, The Kansas Medical Society CME Committee is interested in an active forum for the sharing of CME accreditation information among all the state medical societies; and

WHEREAS, The Kansas Medical Society CME Committee is concerned with the present turmoil surrounding the withdrawl of the AMA from the Liaison Committee on Continuing Medical Education and the subsequent accreditation activities by both organizations, therefore be it

Resolved, That the AMA instruct the Department of Continuing Medical Evaluation to provide regular opportunities for state CME chairmen and staff to meet and interact with AMA continuing medical education staff at the annual AMA/CME conference; and be it further

Resolved, That the AMA be encouraged to explore all possible avenues for the establishment of one national accrediting body.

RESOLUTION NO. 80-5

Kansas Voluntary Effort

Not adopted.

RESOLUTION NO. 80-6

Voluntary Effort

WHEREAS, The legislature will look at prospective hospital rate and budget review during the coming legislative session; and

WHEREAS, There are a variety of different state mechanisms already in existence; some voluntary (Indiana), some legislated (Maryland, Washington State) with some questions about amounts actually saved, and some whose power is being challenged in the courts; and

WHEREAS, The voluntary effort on cost containment has restrained the amount of increase in health care costs and has the approval of physicians, hospital and government; and

WHEREAS, The voluntary cost containment effort encompasses a much wider scope than the hospital rates in its combined efforts; therefore be it

Resolved, That the Kansas Medical Society endorses the Kansas Voluntary Effort for Cost Containment and opposes any type of legislated hospital rate review.

RESOLUTION NO. 80-7

Kansas Hospital Prospective Rate Review

Not adopted.

RESOLUTION NO. 80-8

Legislated Hospital Rate Review

Not adopted.

RESOLUTION NO. 80-9

Principles of Medical Ethics

Resolved, That the Kansas delegates to the AMA House of Delegates be instructed to vote in favor of adoption of a revised version of the Principles of Medical Ethics if necessary.

RESOLUTION NO. 80-10

Faculty Position in Adolescent Medicine

WHEREAS, The health problems of the adolescent age group are unique and require a special expertise of the professional; and

WHEREAS, Training in adolescent medicine as a discipline is lacking at the University of Kansas School of Medicine; and

WHEREAS, The health problems of the adolescent create social problems for the individual and society such as unwanted pregnancy and drug addiction; therefore be it

Resolved, That the Kansas Medical Society recommends additional training in adolescent medicine at the University of Kansas School of Medicine.

RESOLUTION NO. 80-11

Periodic Health Assessment

WHEREAS, Many of the school districts in Kansas do not include health assessment as an essential component of preparation for school attendance; and

WHEREAS, Those school districts which do include assessment find it difficult to maintain a health assessment component in their program without legislative endorsement; and

WHEREAS, Early recognition and/or prevention of health problems and handicapping conditions allow for more effective intervention at less cost; and

WHEREAS, Health assessment must meet standards of quality and provide for proper follow-up; therefore be it

Resolved, That the Kansas Medical Society endorses periodic health assessment by physicians as an essential component of the evaluation of a child preparing for and attending school.

RESOLUTION NO. 80-12

Pregnancy Prevention Services for Minors

WHEREAS, The incidence of pregnancy among minors is increasing at an alarming rate in Kansas; and

WHEREAS, The provision of information and services to minors on pregnancy prevention under the present system has been largely unsuccessful; therefore be it

Resolved, That the Kansas Medical Society go on record favoring the availability of pregnancy prevention information and services to minors by physicians licensed to practice medicine and surgery; and be it further

Resolved, That such services may be provided to minors without the consent of any parent, guardian or other person having custody of such minor; and be it further

Resolved, That the Kansas Medical Society Legislative Committee be directed to draft appropriate legislation for submission to the Kansas Legislature providing that no civil or criminal liability shall be incurred for providing such pregnancy prevention information and services to minors without the consent of any parent, guardian, or other person having custody of such minor.

RESOLUTION NO. 80-13*

Refunds and One-half Year Provisions

WHEREAS, Requests have been received for refunds of membership dues; and

WHEREAS, Requests have been received for one-half year memberships; and

WHEREAS, There are currently no provisions in the bylaws allowing dues refunds or one-half year memberships; therefore be it

Resolved, That the bylaws be amended by deleting from Section 1.71

If leave of absence is taken after the annual dues are paid, they are not refunded.

and be it further

Resolved, That new Section 1.8 and 1.81 be added as follows:

1.8 Dues-Refund
Refunds of one-half (½) memberships dues will be considered on an individual basis upon application to the Executive Committee prior to July 1.

1.81 One-half Year Memberships
One-half year memberships will be considered on an individual basis upon application to the Executive Committee.

RESOLUTION NO. 80-14

Medicaid Reimbursement Policy

WHEREAS, The Medicaid program was designed to provide available medical care of high quality to patients who were unable to pay for such care privately; and

WHEREAS, Payment to medical providers is designated by law to be on the basis of reasonable charges; and

WHEREAS, The Kansas Department of Social and Rehabilitation Services currently reimburses providers at the 50th percentile of their 1975 charges; and

WHEREAS, This level of reimbursement has forced providers to drop out of the program, reducing the quality and availability of services; therefore be it

Resolved, That a system of reasonable compensation for medical providers be established based on current usual and customary fees; and be it further

Resolved, That the Secretary of the Department of Social and Rehabilitation Services be encouraged to comply with K.S.A. 39-708c(x), requiring annual review of the payment schedule; and be it further

Resolved, That the Kansas Medical Society Executive Committee take appropriate action by whatever means they find appropriate to achieve the above goals.

RESOLUTION NO. 80-15

Kansas Medicare-Medicaid Physician Reimbursement

Not adopted.

RESOLUTION NO. 80-16

Demography of Kansas Physicians — Long Range

WHEREAS, The annual number of graduates of American medical schools has increased by 50% within the last decade, during which time the population of the United States has been increasing at the rate of approximately 1.0% per year; and

WHEREAS, Projecting these numbers 10 and 20 years into the future suggests a disproportion in the physician/population ratio; therefore be it

Resolved, That the Kansas Medical Society direct the appropriate committee to undertake study of long term medical school enrollment requirements, and to prepare recommendations for the Kansas Medical Society, the Kansas State Legislature, and the University of Kansas School of Medicine.

RESOLUTION NO. 80-17

Health Care Stabilization Fund

Not adopted. Referred for study.

WHEREAS, The Health Care Stabilization Fund has no provision to equate the excessive charges made to the original health care providers as compared with the lower charges being made to the subsequent entrants into the Fund; and

WHEREAS, No provision is made in the Fund for the return of investment to the participant after the termination of obligations by the Fund; therefore be it

Resolved, That the Kansas Medical Society, along with other participants in the Health Care Stabilization Fund, seek legislative or regulatory changes to provide individual accounting such as an insurance fund in order to: (a) provide each participant credit for his contribution and current value of his shares; (b) charge subsequent entrants comparable amounts consistent with his risk class; (c) repay participants at the termination of obligations of the Fund for that participant for his then current share of the Fund.

SUBSTITUTE RESOLUTION NO. 80-18

HCFA Medicare Program

Resolved, That the Kansas Medical Society reaffirm its support for the concept of private fee for service practice for hospital-based physicians; and be it further

Resolved, That the Kansas Medical Society oppose the HCFA Regulations relating to uniform implementation; and be it further

Resolved, That copies of this resolution be forwarded to the Kansas Congressional Delegation, Governor, Kansas Legislature, and the Health Care Financing Administration.

RESOLUTION NO. 80-19

Physician-Hospital Relationships

Resolved, That the Kansas Medical Society initiate appropriate action to establish, on a cooperative basis, an official liaison group to be composed of individuals representing the KMS, KHA hospital Boards of Trustees and others as deemed appropriate to work cooperatively and harmoniously in preserving the private practice of medicine and health care delivery in the State of Kansas.

RESOLUTION NO. 80-20

Laboratories

WHEREAS, There are in existence, in various areas of Kansas, laboratories that are performing lab tests and providing lab services where there is no physician involvement or supervision; and

WHEREAS, These labs do not come under the Medicare Rules and Regulations and thus are able to operate with little or no review or restriction; and

WHEREAS, These types of operations may not be in the best interest of the public and because their operations reflect unfavorably on reputable laboratories, as well as the entire medical profession; and

WHEREAS, These types of questionable operations encourage the promulgation of needless rules and regulations; therefore be it

Resolved, That the Kansas Medical Society Council meet with representatives of the State Department of Environment's Office of Laboratories and the Kansas State Board of Healing Arts for the purpose of discussing mechanisms to control the operation of laboratories operating in Kansas which are outside the jurisdiction of current licensing and review agencies or organizations.

RESOLUTION NO. 80-21

Physical Therapy

WHEREAS, Athletic and exercising oriented groups are considering the use of ultra-sound, diathermy, and other physical therapy modalities in connection with their physical fitness programs; and

WHEREAS, Few, if any, of the staff from these groups are Registered Physical Therapists or Physical Therapy Assistants; and

WHEREAS, The current revised Rules and Regulations of the Kansas State Board of Healing Arts' Physical Therapy Committee do not preclude the use of ultra-sound and diathermia by persons who are not Registered Physical Therapists or Physical Therapy Assistants; and

WHEREAS, There may be a significant medical risk to persons receiving ultra-sound and diathermy treatment from persons inadequately trained; therefore be it

Resolved, That the KMS bring this matter to the attention of the Kansas State Board of Healing Arts' Physical Therapy Committee and request that, in the public's interest and safety, consideration be given to amending the existing Physical Therapy Rules and Regulations setting forth those modalities which may be performed only by or under the direction of physicians and/or Physical Therapists.

RESOLUTION NO. 80-22

Definition: Practice of Medicine

WHEREAS, Since there appears to be a continued proliferation of the paramedical groups and services in the State of Kansas; and

WHEREAS, The majority of these services offered to the public by members of these groups are, or border on, the practice of medicine; and

WHEREAS, The majority of these groups do not come under the jurisdiction of the official licensing or accreditation bodies; and

WHEREAS, The local District Attorney's offices are therefore responsible for determining whether or not such groups or individuals are illegally practicing medicine for which no definition exists; therefore be it

Resolved, That the Council of the Kansas Medical Society be directed to evaluate in cooperation with the Kansas State Board of Healing Arts the advantages and disadvantages of defining the practice of medicine.

RESOLUTION NO. 80-23

Advanced Registered Nurse Practitioner: Roles and Responsibilities

Not adopted.

RESOLUTION NO. 80-24

Advanced Registered Nurse Practitioners — Policy Statement

WHEREAS, The 1978 Legislature approved enabling legislation creating the Advanced Registered Nurse Practitioner; and

WHEREAS, Rules and regulations governing the role of the ARNP are currently being developed by the Kansas State Board of Nursing; therefore be it

Resolved, That the Kansas Medical Society adopt the following statement as its official policy regarding ARNP: The Kansas Medical Society supports the basic concepts of the Advanced Registered Nurse Practitioner and recognizes that an appropriately educated nurse can competently perform selected delegated medical tasks traditionally performed personally by the physician; however, the provision of these services remains the responsibility of the physician licensed to practice medicine and surgery; and the ARNP functions as a member of a physician-directed health care team; and be it further

Resolved, That a copy of this resolution be forwarded to the President of the Kansas State Board of Nursing.

RESOLUTION NO. 80-25

Advanced Registered Nurse Practitioners — Teaching Programs

WHEREAS, Registered nurses in Kansas are currently being trained to function in expanded roles as ARNPs; and

WHEREAS, The ARNP curriculum includes the teaching of medical subjects beyond those included in the usual Registered Nurse curriculum; and

WHEREAS, To assure that the ARNP is adequately trained to perform in this expanded role; therefore be it

Resolved, That physicians be involved in developing and teaching those medical segments of the ARNP curriculum which are beyond the traditional Registered Nurse curriculum; and be it further

Resolved, That a copy of this resolution be forwarded to the President of the Kansas State Board of Nursing.



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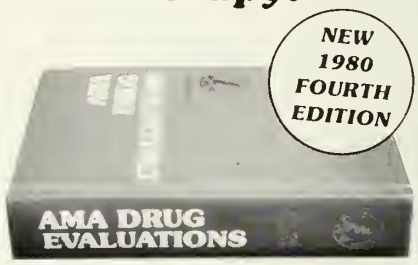
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RESOLUTION NO. 80-26

M.D. — Nurse Liaison Committee

WHEREAS, The roles, functions, and responsibilities of members of the medical profession and the nursing profession are becoming more complex and of mutual concern; and

WHEREAS, It is imperative that there be mutual understanding and respect among the members of these professions so as to assure quality patient care; therefore be it

Resolved, That the Kansas Medical Society initiate appropriate action to bring about the development of an active ongoing MD-Nurse Liaison Committee for the purpose of furthering cooperative relationships and mutual understandings; and be it further

Resolved, That the membership of this committee be composed of practicing physicians, nurses from the various nursing groups, and representatives from the State Boards of Nursing and Healing Arts as well as other appropriate individuals.

SUBSTITUTE RESOLUTION NO. 80-27

Ophthalmology Curriculum at the University of Kansas School of Medicine

WHEREAS, The Ophthalmology curriculum at the University of Kansas School of Medicine has been reduced; and

WHEREAS, Attempts to improve this curriculum have been unsuccessful; and

WHEREAS, Other departments have encountered similar cuts in graduate curricula, such as the Neurology Department which no longer has any required hours; therefore be it

Resolved, That the Kansas Medical Society encourage the Curriculum Committee of the University of Kansas School of Medicine to increase the training of medical students in Ophthalmology and other subspecialties.

RESOLUTION NO. 80-28

Nuclear Low Level Waste Depositories

WHEREAS, The use of tracer radioactive materials used for the diagnosis and treatment of disease has had a profound effect on the quality and length of life for the citizens of the State of Kansas by providing valuable and often life-saving information about cancer, heart, renal, lung, and bone diseases through accurate, safe, and cost efficient methods; and

WHEREAS, The use of radioactive cobalt and other such substances have unequivocally proven their value to treat many cancers for cure or palliation to improve the quality of life; and

WHEREAS, No medical radioactive material is produced in our state making us totally dependent on out-of-state manufacturers for these materials; and

WHEREAS, Most all of the diagnostic and therapeutic material used in the state is of such a minute level as to be barely above naturally occurring radiation that the NRC (U.S. Nuclear Regulatory Commission) describes it as "exceptionally low level" and is recommending the adoption of a "deminimus" standard for handling it; and

WHEREAS, Unless proper "low level" radioactive waste depositories to handle the by-product waste generated by hospitals or other medical facilities performing diagnostic tests, cancer therapy, or medical research are established free of the whims and political moods of the governor and legislature, we may soon find ourselves without any medical radioactive substances to help our patients in our great state; and

WHEREAS, The licensing, inspection, and regulation of medical and industrial radioactive material use and waste disposal is currently performed by the Kansas Radiation Control section of the Department of Health and Environment as specified by Kansas State Laws and the U.S. Nuclear Regulatory Commission; therefore be it

Resolved, That radioactive wastes generated by the practice of Nuclear Medicine, Radiotherapy, in medical research laboratories, and educational institutions be exempted from the approval by the Governor of the State of Kansas and the legislative body of the State of Kansas. Also exempt should be the radioactive waste generated in the production of radionuclides and radiocompounds for the practice of Nuclear Medicine.

RESOLUTION NO. 80-29

Journal Advertising

Resolved, That the House of Delegates establish the Kansas Medical Society legislative proposal on advertising (SB #542 as introduced) as its policy for accepting advertising in the Journal of the Kansas Medical Society.

RESOLUTION NO. 80-30

Hospital Rate Review

Resolved, That resolution 79-6 be rescinded.

RESOLUTION NO. 80-31

Medico-Legal Autopsies

Not adopted. Referred for study.

Resolved, That in the interest of promoting the most scientifically accurate collection of data which may subsequently have a significant or decisive role in the judicial process, the Kansas Medical Society recommends that medico-legal autopsies be performed only by or under the direction of physicians trained in the discipline of forensic pathology or anatomic pathology; and be it further

Resolved, That copies of this resolution be disseminated to all the Judges of the District Courts of Kansas; and be it further

Resolved, That copies of this resolution be disseminated to the Kansas Legislature.

RESOLUTION NO. 80-32

1980 Commendation/1981 Invitation

WHEREAS, The 121st Annual Session of the Kansas Medical Society is only seconds away from being completed; and

WHEREAS, The joint effort of the Johnson and Wyandotte County Medical Societies has been extremely well done; therefore be it

Resolved, That the Kansas Medical Society thank both Johnson and Wyandotte counties for a well organized and successful meeting; and be it further

Resolved, That this resolution serve as an invitation by the Saline County Medical Society to welcome the 122nd Annual Session of the Kansas Medical Society to Salina in 1981.

(Continued from page 305)



At the President's Banquet, Phoebe and Phil Godwin take an optimistic and courageous view of the responsibilities they will share during his year as President of the Kansas Medical Society—



—while Bill Walker, Treasurer of the KMS, inspects the corsages worn by Sally Goering and his wife, Alleein.

(As usual, the *Journal* is indebted to Dr. Jack Cooper for his photographic record of the meeting, no mean contribution considering the cost of film these days.)

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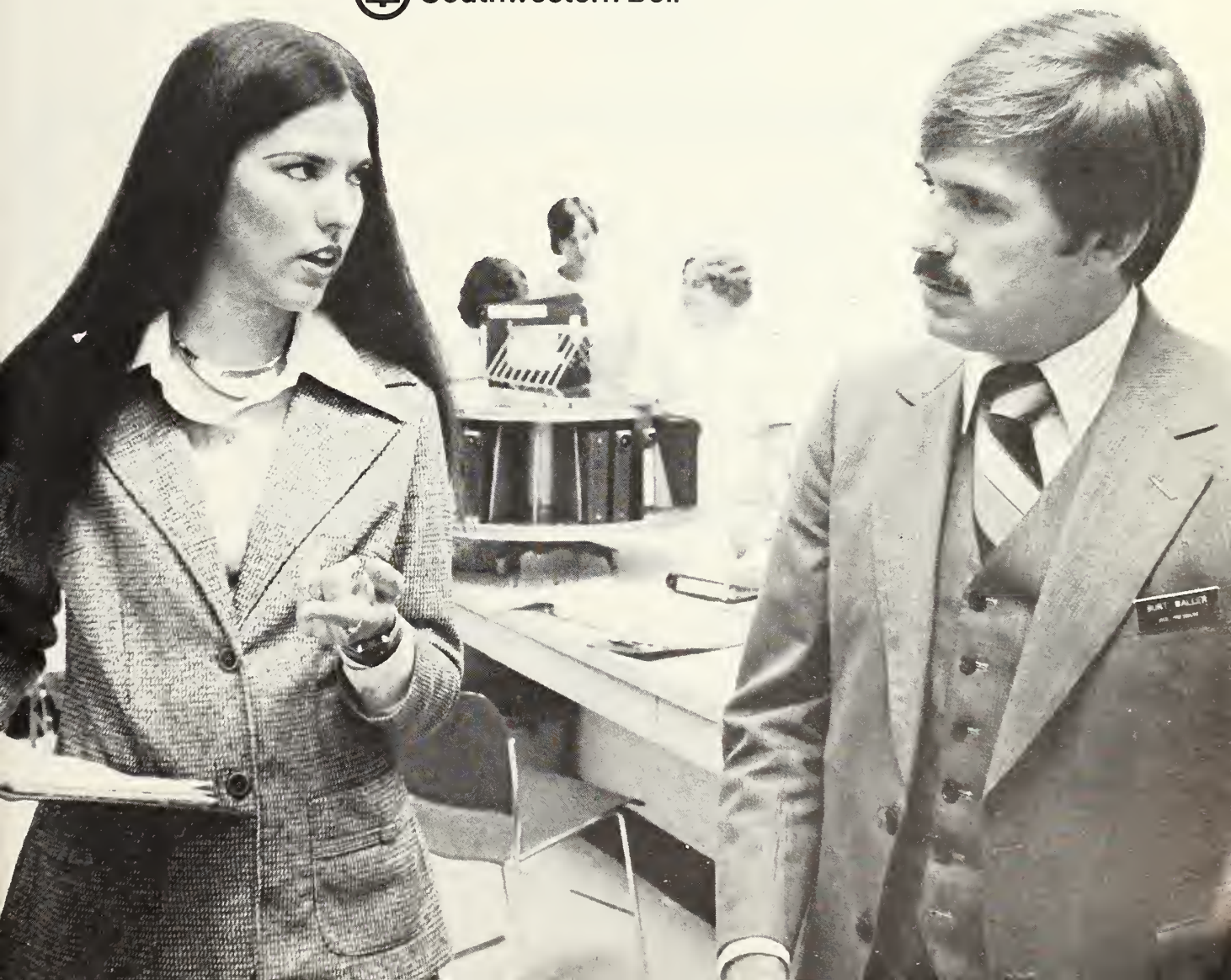
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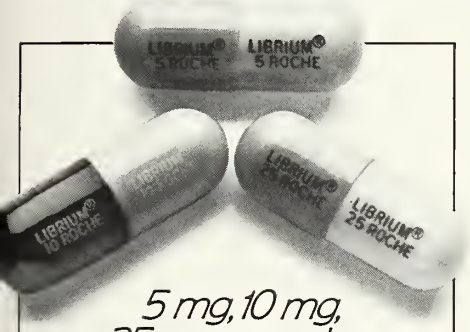
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Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium* (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose* packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs* (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable

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Information for Authors

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Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.



A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC®

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbrevieted Summary

***INDICATIONS**: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FOA has classified the indications as follows.

"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenitel melformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diezepam) during the first trimester of pregnency hes been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost elways be evoided. The possibility that a woman of child-bearing potential may be pregnent et the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placentet barrier. It is present both in umbilical-cord blood at or near maternal plasma levels end in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk es compered to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous adminstration of meprobamate are reported; most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

Wyeth Laboratories
Philadelphia, Pa. 19101

FOR MODERATE PAIN

A therapeutic dose of acetaminophen in *one* tablet

A therapeutic dose of two complementary analgesics

The convenience and economy of a dosage schedule of one tablet, every four hours as needed



WHY NOT WYGESIC® (IV)

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbreviated Summery

INDICATION: For the relief of mild-to-moderate pain

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Menegement of Overdosege**)

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g driving a car or operating machinery Patients should be cautioned accordingly

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenedrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances The chronic ingestion of propoxyphene in doses over 800 mg per dey has caused toxic psychoses and convulsions Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Wernings**) Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine

MANAGEMENT OF OVERDOSAGE: SYMPTOMS The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage end include respiratory depression (a decrease in respiretory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse In eddition to these characteristics, which are reversed by narcotic entagonists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay ot cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases ot severe poisoning Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred

Symptoms ot massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours However, early recognition may be difficult since early symptoms may be mild and nonspecitic Evidence of liver damage is usually delayed After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation detects, hypoglycemia, encephalopathy, coma, and death may follow Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more ot acetaminophen may produce hepatotoxicity A 13-gram dose has reportedly been fatal.

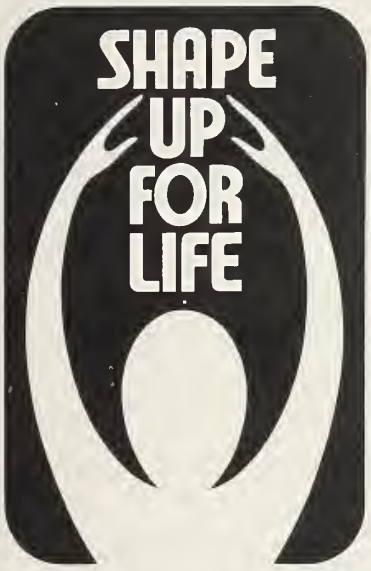
TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision ot e patent airway and institution of assisted or controlled ventilation The nercotic antagonists, naloxone, nalorphine, and levallorphen, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these entagonists should be administered, preterably IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant fo control seizures Analeptic drugs (e.g ceffeine or amphetamine) should not be used because of their tendency to precipitate convulsions

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated Gastric lavage may be helpful Activated charcoal can absorb a significant amount ot ingested propoxyphene Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed Copious gastric lavage and/or induction ot emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload It has been reported that mercaptamine (cysteamine) or other thiof compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity Seriel hepatic enzyme determinations are elso recommended

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Philadelphia, Pa 19101



American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

AUXILIARY NEWS

An Open Letter to Kansas Physicians

The Kansas Medical Auxiliary recently convened the summer Board of Directors meeting and workshop in Overland Park. Our membership was well represented as the State Chairmen presented their programs and County Auxiliary Presidents shared their projects.

Dr. Phil Godwin, KMS President, spoke of his plan for Auxiliary members to serve on some KMS committees. We hope to fill these positions soon. Auxiliary members currently serve on the Impaired Physician and Legislative committees.

Jerry Slaughter discussed the legislative process and urged Auxiliary members to become better acquainted with their representatives. He stressed the benefits of presenting medicine's point of view in advance of the busy election time.

KMS Auxiliary delegates to the AMA Auxiliary convention will be Mmes. Robert Moore, Frank Bichlmeier, Kermit Wedel, David Laury, Chester Young (Past National President), and Clair Cavanaugh (National AMAERF Chairman 1980-81). Activities will begin on Saturday, July 19, with conferences on legislation, health, membership, and AMAERF.

The Kansas Body Pollution Learning Center is being displayed at various health conferences. At least seven states have constructed Learning Centers from our instruction kit. We are pleased that this project — which originated with the Wyandotte County Auxiliary — is being widely utilized.

Body Pollution Centers are used in grade schools to teach fourth and fifth graders the facts about nutrition, alcohol, smoking, drugs, and mental health. Students learn through film strips, reading, group games, and individual activities. Two traveling centers are available for use in local schools in Kansas. Mrs. Richard Glover, Newton, schedules these units. Several counties have their own units or are in the process of building them. The Learning Centers are popular both with students and teachers, and we hope additional County Auxiliaries will construct their own units for local use. It has proven to be an excellent public relations program.

Sincerely,
Evelyn Huff
President
Kansas Medical Society Auxiliary

Symptom: rising administrative costs

Prescription: cost-containing ideas from Southwestern Bell

He's Vice-President for growing Methodist Hospital in Dallas. She's a health care Account Executive for Southwestern Bell.

And whether she or one of her health care team members is reviewing procedures in the admissions area or consulting with hospital administrators, she's one hard-working "problem solver."

Able to dig into your health care facility, study it carefully and emerge with new ideas about how you can use communications to increase efficiencies and contain costs.

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The results — emergency room attendants receive vital information from doctors faster than ever, patients can easily contact admissions clerks handling their files and the hospital's telephone attendant requirements were cut in half.

To find out how a Southwestern Bell Account Executive can help you solve your health care problems and contain costs, call us today.

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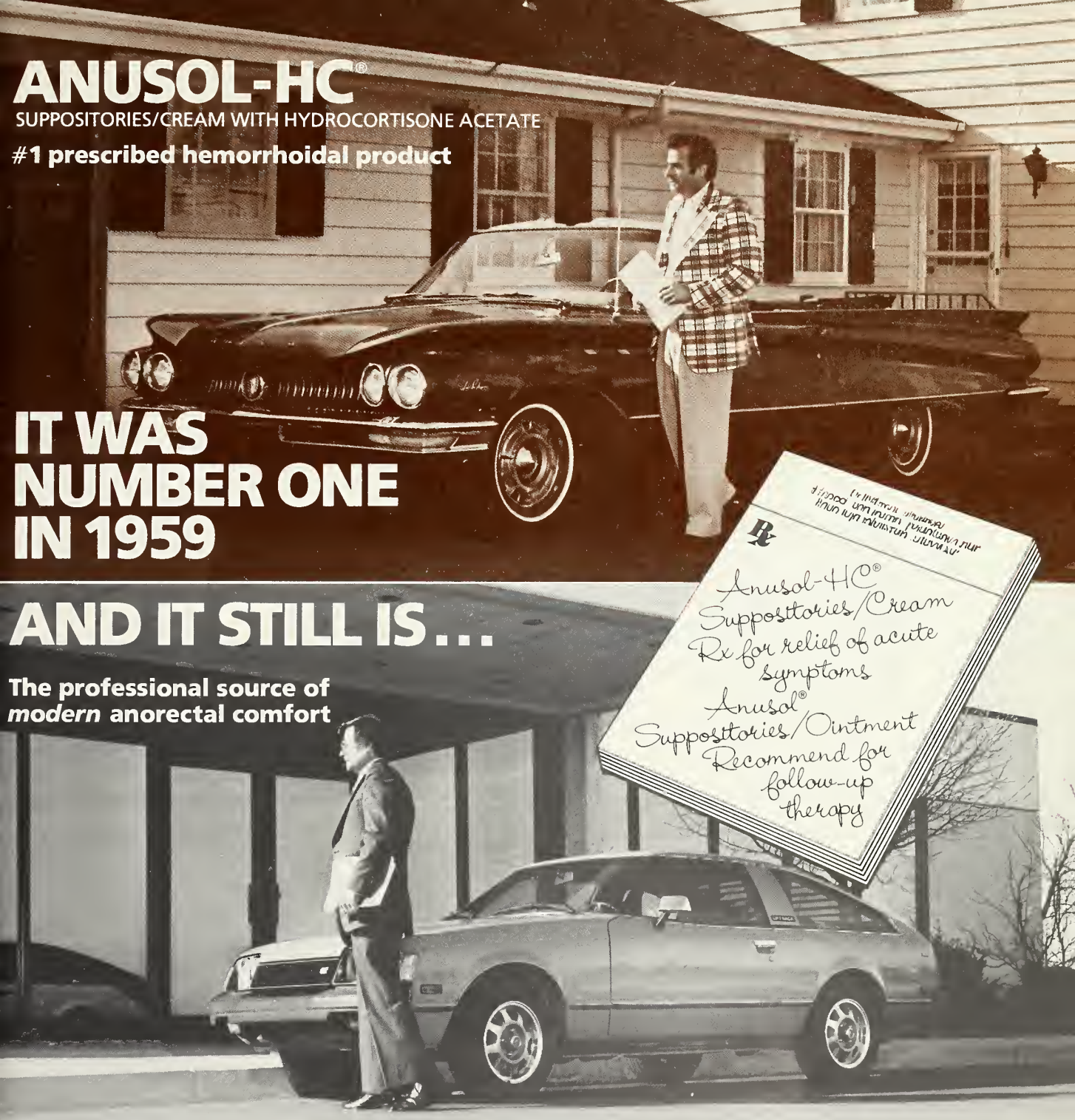
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#1 prescribed hemorrhoidal product

IT WAS NUMBER ONE IN 1959

AND IT STILL IS...

The professional source of *modern* anorectal comfort



ANUSOL-HC® SUPPOSITORIES
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ANUSOL-HC® CREAM
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Caution: Federal law prohibits dispensing without prescription.
Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

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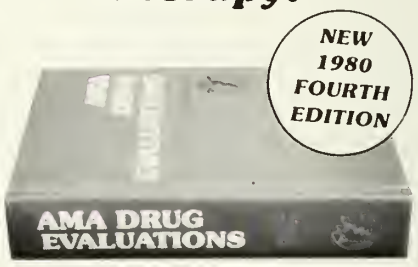
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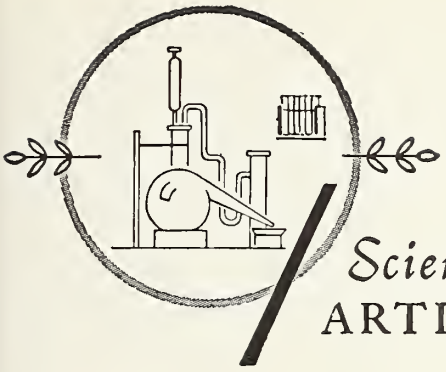
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Gynecological Trauma

Non-Obstetrical Vulvar, Urethral, and Vaginal Injuries

JEROME S. MENAKER, M.D.,* *Wichita*

Trauma to the female external genitalia is fairly common but is often neglected in gynecologic, surgical, and family practice training. These injuries, while often minor, may be life threatening and occasionally fatal. Most such injuries are accidental, but may result from forcible coitus. Standard texts and current literature are briefly reviewed, and some illustrative cases are reported.

Trauma to the female external genitalia is a fairly common occurrence but is often slighted or completely neglected in gynecology, surgery, urology, and family practice training programs. A physician's initial exposure to such problems is often an encounter in the emergency room or on the surgical floor with a patient suffering from one of the more serious injuries. Formal discussion, case studies, and clinical conferences on the subject are rare. With increasing emphasis on girls' participation in sports programs from an early age and the increased reports of rape, trauma to the vulva, vagina, and urethra is also increasing.

This subject has been inadequately addressed in current standard texts in gynecology and in recent specialty literature. For example, Novak's *Textbook of Gynecology*[1] makes no mention of the subject; and Parson-Sommers' *Gynecology*,[2] a two-volume, 1660-page text, devotes only three paragraphs to the subject. Jeffcote's *Principles of Gynecology*[3] allots less than two pages to non-obstetric trauma of the external genitalia. A survey of recent literature revealed only seven references, four of which were case reports contained in "Letters to the Editor." These included three references to water skiing and one to cross-country skiing.

Tweedale[4] and Edington[5] report cases of injuries inflicted by forcible water spray resulting from falls while water skiing at high speeds. These reports were all similar in that each patient was wearing a standard swim suit, fell with thighs abducted, and was pulled from the water bleeding profusely and in danger of shock. In each of the three cases, there was a deep vaginal laceration with arterial bleeding. One patient required transfusion of 1500 cc of whole blood. One woman had a severely bruised anus and rectum, and continued to drip water for some time from what was, in effect, a forcible enema and douche. In each case, the edema and ecchymosis continued following treatment. In one patient the vulva was reported to be black one week following injury. McCarthy[6] and Morton[7] report two cases of severe vulvar injury due to the direct blow of a water ski. Lyons and Porter[8] describe a vulvar hematoma resulting from a fall during cross-country skiing. The 18-year-old female fell, then skied cross-country for

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24 km to her home, after which she required hospitalization for one week with bed rest and heat until the hematoma subsided. No attempt at evacuation of the hematoma was described.

Rape — being reported with increasing frequency — often causes severe trauma to the female genitalia. Bruising and laceration of the vulva, vagina, and anus are fairly common. It is surprising that in many cases these injuries are objectively and somatically minor. Very young girls and older women are more prone to trauma of the genitalia as a result of rape, due to the small size and immaturity of organs in the former and the inelasticity of tissues in the latter. However, even the recent organized rape of Bangladesh females of all ages by troops of the Pakistani army resulted in few major injuries to vulva and vagina; this was noted by American volunteer physicians who went to Bangladesh on a mercy mission to perform abortions on these women.

It is interesting, then, that severe vaginal lacerations sometimes result from sexual activity occurring with mutual consent but often involving ingestion of alcohol. Such coital injury is more common after total hysterectomy — whether vaginal or abdominal — because of the shortened vaginal vault.

Trauma has been reported in cases of lesbian sexual activity and also during masturbatory activity with a variety of objects and foreign bodies including vibrators.

Urethral injury due to trauma is more common in the young girl because the urethra is shorter, smaller, and closer to the external area of the vestibule. It is also less likely to be promptly reported by the young girl; older women will more readily complain of the trauma. Morton[9] notes that injuries to the fourchette, labia, or urethra may be a cause of acute urinary retention in young girls. She records two cases of injury resulting in severe urinary retention that at first resulted in confused diagnosis. The first was a 4-year-old injured by the horizontal bar on a boy's bicycle and the other a 2-year-old who straddled some playground equipment. Each case presented with acute urinary retention and a urinary bladder at the level of the umbilicus. There was no history of trauma, nor did the first questioning reveal the source of the trouble. In each case, superficial tears of the fourchette and labia were revealed by examining the child with the labia spread. An application of an anesthetic cream was used with each desire to void and the child was encouraged to micturate while sitting in a warm tub of water. In each case, catheterization was avoided.

This type of urine retention has not been frequently described and is easily overlooked. Injury to the perineum or vulva can stretch and tear the mucosa. Just a few drops of urine can cause intense pain and spasm. This combination inhibits voiding so that complete retention may then ensue. A similar situation often occurs with herpes lesions of the vulva.

Williams[10] wrote of problems of female children with ruptured urethras. As previously noted, except in pelvic fractures, this injury is more common in young girls. The injury will almost always extend to the vagina. Heller[11] suggested that this type of injury may be overlooked in the differential diagnosis of vaginal bleeding in girls under the age of 10 years. The source of external bleeding may be deceptive in that tears in the wall may bleed into the vagina and result in minimal external blood loss. Examination under anesthesia may be necessary, especially if the hymen is lacerated. Four of the 51 cases of abnormal bleeding reported in young girls were due to injury resulting from falls; fifteen were due to precocious puberty; and the rest due to local lesions, including malignancies.

In the past, another major cause of genital injuries in the female was crude, clumsy, or brutal attempts at abortion by amateurs or by the woman herself. Under present law, such injuries are markedly decreased.

Gunshot wounds of the female genitalia are apparently infrequent; a search of both texts and literature failed to reveal a single recorded case. One such incident will be included in the case reports below.

The diagnosis of female genital injuries is often obvious, but as in all medicine, may require a detailed history and meticulous physical examination. In the young, this may include examination under anesthesia.

Treatment in many cases will consist of the application of ice packs with pressure followed by warm compresses and sitz baths. Many of these lacerations heal quickly and spontaneously and do not require suturing. Hematomata may sometimes require evacuation and pressure dressing. The use of oxidized cellulose — such as oxycell cotton with iodoform packs — may be required in badly traumatized tissue that acts like wet blotting paper and will not hold a suture. Arterial bleeding requires meticulous ligation with suturing of the overlying mucosa. Injuries to the urethra may require an indwelling catheter for from five to seven days. Rarely is there an indication for tetanus antitoxin or antibiotics. In cases of severe bleeding, blood replacement is appropriate and may be life saving.

The use of proteolytic enzymes such as bromelains (Ananase) or trypsin-chymotrypsin (Chymoral)

may be helpful in facilitating absorption of hematomata, ecchymosis and edema, and may speed healing and tissue repair.

Abdominal exploration to staunch intra-peritoneal bleeding through the vaginal vault or broad ligament is possible, but fortunately is rarely necessary.

Many such injuries can be prevented. Little girls should avoid riding boys' bicycles with horizontal bar. Women engaged in active sports should wear protective clothing. Water skiing should be done at a speed commensurate with the skier's ability. The skier should wear cut-off jeans or a short wet suit. Newly married, post surgical, and post menopausal patients should be cautioned — but not frightened — as to the possibility of sexual injury. Good advice is often to make love as porcupines do — very carefully.

Case Reports

Case One: A 7-year-old female was brought into the emergency room with a 5-cm hematoma of the right labium major with ecchymosis extending above the inguinal ligament. The child had been doing "wheelies" on a boy's bicycle and fell across the cycle bar. The hematoma was evacuated and the area packed with oxycell cotton.

Case Two: An 11-year-old female was admitted with a long tear in the labium minor and a badly contused urethra incurred as the result of a fall while walking on a picket fence. She required ice packs for 24 hours; then heat and bed rest for five days with urethral catheter.

Case Three: A 20-year-old female was brought to the emergency room in profound shock. She had been at a sex party with three males and one other female, all inebriated. While literally being chased around the room nude, she slipped and fell on the rung of a rocking chair. Treatment included suturing of a bleeding artery, vaginal packing, a urethral catheter, and three units of whole blood.

Case Four: A 65-year-old female presented with a stellate laceration of the vault of the vagina. She had been sexually inactive for 18 years; subsequent coitus resulted in a jagged tear in the posterior fornix that defied suturing but responded to a vaginal pack applied for 48 hours.

Case Five: A 19-year-old newly married female presented, near shock, pale and anemic, and with a hemoglobin of 7 gms as a result of coital trauma. Her husband was leaving the following day for an indeterminate time, and they had intercourse five times during the night. On the sixth attempt, the right vaginal fornix was lacerated with two arterial bleeders that required ligation. Three units of packed red cells were given to correct acute anemia.

Case Six: A 38-year-old unmarried female was seen with the complaint of urinary incontinence. She had been masturbating with a succession of articles including a swizzle stick, a pencil, and a toy rubber cigar. The hymen was rigid and intact, but the urethra was wide, gaping, and partially prolapsed. The patient was treated with a minimal basic lesson in female anatomy and plication of the urethra.

Case Seven: A 40-year-old married female was brought to the emergency room in an ambulance with her entire perineum shot away by a shotgun blast. Her husband had just returned from a hunting trip to find his wife bending over a bath tub, and he playfully "goosed" her with a muzzle of the shotgun. The gun discharged, tearing away the anus, vagina, vulva, and part of the bladder. Following extensive surgery, the patient had a cloaca, colostomy, and an ileal bladder.

Summary

Several types of non-obstetrical external genital injury in females have been reviewed and several case reports presented. It is suggested that therapy be limited to minimum adequate procedures. Often cold or hot packs and pressure will accomplish a cure. When necessary, prompt ligation of bleeders with suturing and blood replacement are appropriate. In the very young female, a questioning attitude must be maintained and physical examination comprehensively conducted because the history may be difficult or impossible to elicit.

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Hepatic Abscess

Radioisotope Scanning and Ultrasound Imaging

REZA P. RASSA, M.D., *Salina*

AUTOPSY FINDINGS indicate that prior to 1966, a correct antemortem diagnosis of hepatic abscess was made in only 19 per cent of all cases. Since that time, the widespread availability of radioisotope scanning (scintiscan), and more recently, the gray scale ultrasound imaging (sonogram) have improved the situation to a point where today a correct diagnosis is made in more than 90 per cent of these patients at the time of clinical presentation.[1, 2]

General Considerations

Hepatic abscesses are an infrequent but life threatening condition. The lesions are caused by two agents — amebic and bacterial. Rarely, abscesses due to fungi or Echinoccus have been reported. The incidence of pyogenic abscess is much greater than that of amebic (80% *vs* 8-10%).[1, 3, 4] The organisms most frequently seen are bacilli (Escherichia coli), cocci (Streptococci), and anerobic organisms (Bacteroides funduliformis).[3]

The abscesses may be single or multiple. The single abscess occurs more commonly in the right lobe. The left lobe has been reported to be involved in less than 5 per cent of all cases. One series of 39 cases included 24 in the right lobe, seven in the left, and eight bilateral.[1]

Hepatic abscesses occur in all age groups. In adults, the incidence is greater among males, and the diabetic population appears to be at greater risk than the general population.

Until the mid 1960s, hepatic abscess was associated with poor prognosis and high mortality. Diagnostic difficulty which typically resulted in delayed treatment was substantially responsible for the situation. In the pre-antibiotic era, mortality approached 100 per cent, remaining as high as 30 per cent in 1969. Today, antibiotic therapy is an adjunctive agent of considerable value, but it has not been effective in reducing mortality without surgical drainage, the present day treatment of choice.[1, 3]

There have been no deaths from pyogenic hepatic abscess reported among patients diagnosed at the time of presentation.[5] When fatal cases have occurred, diagnosis was not promptly made, and in each of these patients, abscess was diagnosed only at the time of necropsy.

The most common sources of infection include:

1. Portal pyaemic, secondary to appendicitis, diverticular disease, Crohn's disease, ulcerative colitis, or other inflammatory disease in the portal system;
2. Cholangitis, secondary to calculus or malignant obstruction of the bile ducts. Lesions are generally multiple and prognosis is poor. This is becoming a less common cause;
3. Direct spread from contiguous disease such as cholecystitis, peptic ulcer, subphrenic and perinephric infection;
4. Trauma, either blunt or penetrating;
5. Infarction after embolism or complicating sickle cell disease;
6. Cryptogenic — with the falling incidence of major intra-abdominal infections, this source of abscess is becoming proportionately more common.

In 43 per cent of one series, no primary site of sepsis was found.[3] These abscesses seem to occur after the resolution of an infection in the portal system or as a sequela to intrahepatic thromboembolism and infarction.

The availability and use of radioisotope liver scanning (scintiscan) and gray scale ultrasound imaging (sonogram) have had a significant impact on the diagnosis and treatment of hepatic abscess. Early diagnosis and localization with these techniques have greatly reduced mortality and expedited surgical intervention.

Clinical Features

In the absence of a preceding, obvious cause to suspect hepatic abscess, the only symptom may be the non-specific signs of sepsis. The most constant indicator is fever (95%) ranging from 37.5-40.0 C. The febrile state may be continuous or intermittent, but when present tends to be hectic, particularly in

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the multiple abscesses associated with cholangitis.

Dull, relatively steady abdominal pain is another common presenting complaint. The pain is most often localized in the right upper quadrant or mid epigastrium. The pain may be severe enough to suggest acute abdomen. Inspiration may aggravate the pain due to diaphragmatic irritation, and pain may radiate to the neck or ipsilateral shoulder. About 10 per cent of all patients present with vomiting. Other non-specific symptoms and signs such as malaise, anorexia, nausea, weakness, and weight loss may appear.

The picture is somewhat less obscure when the abscess represents a complication of a preceding primary event. Conditions that may signal hepatic abscess include appendicitis or acute cholecystitis, and surgery with apparent recovery followed by persistent fever, signs of chronic infection, and pain in right upper quadrant.

The most frequent physical finding is an enlarged, tender liver. The enlargement, however, may be superior rather than inferior. About one-third of all patients are jaundiced. Ascites is infrequent, except as a terminal finding.[3, 5] Pleural effusion is relatively common.

Diagnostic Studies

Roentgenographic studies, including scintiscan and sonogram, are the most important diagnostic aids. There are a variety of other protocols which yield collaborative evidence for the diagnosis. Elevation and limitation of motion and the diaphragm and pleural effusion are commonly observed by chest roentgenogram. If the infection is due to a gas forming organism, an air fluid level may be seen.

Biochemical studies reveal a white blood count indicative of leukocytosis with a marked increase in polymorphonuclear leukocytes. Elevated WBC is greater in pyogenic than in amebic abscess. Anemia may be marked in the patient with a large chronic abscess. Liver function tests may be abnormal, particularly in alkaline phosphatase, although it is not uncommon for all to be normal. Elevation of the bilirubin is relatively infrequent. Positive blood cultures may be recorded.[1, 5]

With the exception of the scintiscan and sonogram, none of these procedures are pathognomonic. Scintiscan and sonogram are a direct approach to the lesions of the liver, particularly mass lesions such as tumor, cyst, or abscess. Hepatic abscess in a technetium-99 colloid scintiscan presents as a cold defect area when a single abscess is present, or multiple cold areas when multiple abscesses are present. This finding, in association with the clinical signs, permits a high degree of accuracy in diagnosis of about 90 per cent of cases.[6]

Gray scale sonography further assists in distinguishing between solid and cystic intrahepatic masses. The new generation of ultrasonic equipment is capable of enlarging details of the small scale internal anatomy of tissues in addition to the outline of organs. The sonogram is reported to produce fewer false positives than the scintiscan, and is therefore a valuable tool in equivocal radionuclide studies. For example, a lesion of less than 2 cm can be detected with present sonograms. The degree of diagnostic accuracy in a sonogram is about 88 per cent.[2, 6]

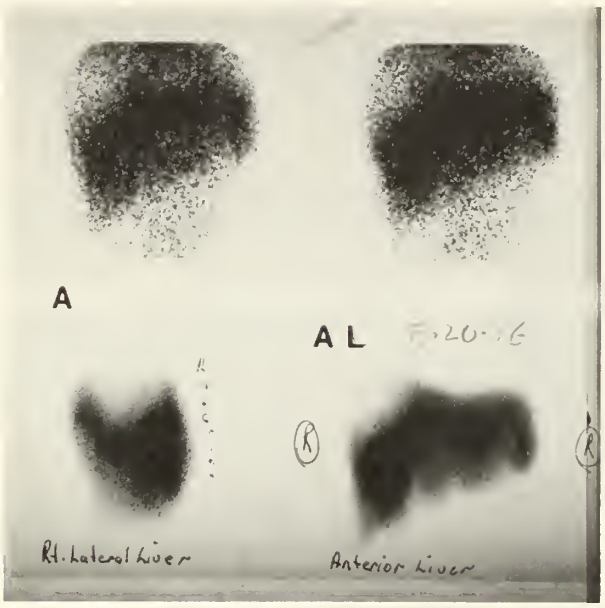
Single or multiple abscess cavities present as echo-free areas by means of return of very low-level echoes, indicating a fluid consistency. The whole liver substance might return high-level echoes consistent with an inflammatory process. The diaphragm may not display, or an echo free area may appear above the diaphragm, indicating pleural fluid. In most instances, the complementary use of scintiscan and sonography techniques provides a better evaluation than either technique used alone.[7]

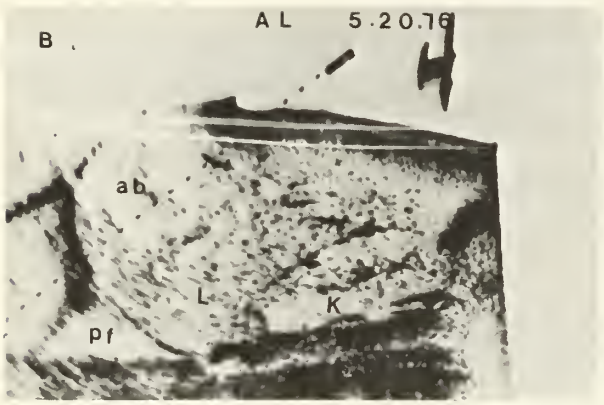
Case Reports

Case One: A 13-year-old Filipino female was admitted to the hospital with a history of abdominal pain, nausea, vomiting, and hectic fever. She had no diarrhea and no history of major illness. A prior physical examination had yielded normal results. The admitting physician's diagnostic impression was either hepatitis or hepatic abscess. Laboratory studies revealed a slight elevation of SGOT (258) and LDH (59). Alkaline phosphatase was 360, and WBC was 21,000. Urinalysis and chest x-rays were normal.

Scintiscan *(Figure 1A)* revealed hepatomegaly as a generalized mottled uptake with a very definite area of decreased uptake within the superior and lateral aspect of the right lobe, suggestive of numerous abscesses and diffuse metastatic disease. Ultrasonography revealed a relatively transonic area measuring 8 x 8 x 9 cm within the right lobe with strong internal echoes *(Figure 1B),* and evidence of pleural effusion. Thoracentesis revealed no amoeba. Febrile agglutinins were positive 1:40 proteus OX-19. Lupus erythematosus preparation was negative, and indirect hemaglutination for amebiasis was normal.

One week after admission, needle aspiration of the liver was performed under fluoroscopy control. The aspirant contained pus, and enterobacter aerogens were determined by culture. The patient was treated





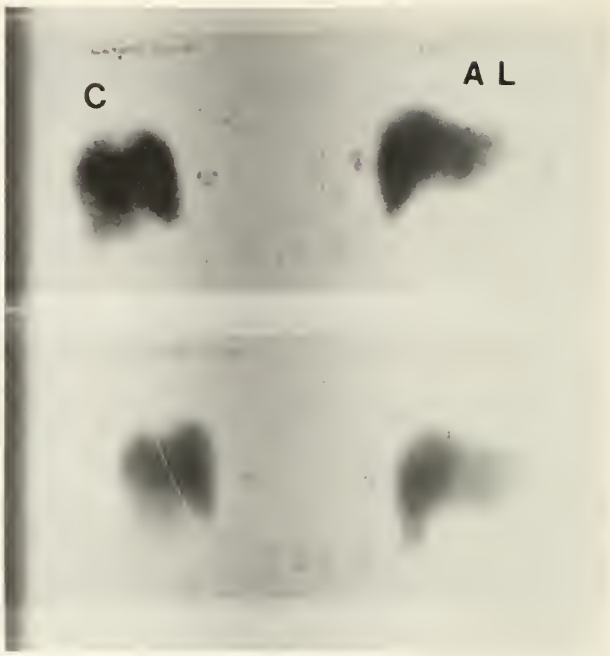


Figure 1. A. Anterior and posterior scintiscan showing enlarged liver with a defect in the superior and lateral aspect of the right lobe. B. Longitudinal ultrasound section of the liver 4 cm right to the midline; demonstrates an irregular area of low level echo (echolucent) irregular echo within it; represents a liver abscess (ab). Note pleural fluid (Pf) as a triangle translucent in the posterior sulcus. C. Follow-up scintiscan showing gradual decrease of the abscess and liver appearing normal in size.

with Garamycin, 40 mg/eight hours intramuscularly, and tetracycline, 250 mg 4 times/day, administered for ten days.

The patient became afebrile and WBC returned to normal. Recovery proceeded without incident, and tenderness in the right upper quadrant lessened. She was discharged on the 20th hospital day. A follow-up scan six weeks after discharge demonstrated a marked decrease in the size of the defect of the right lobe of the liver *(Figure 1C).*

Case Two: A 61-year-old white male was hospitalized with fever, chills, and abdominal pain with right shoulder radiation. The patient was a known diabetic controlled with Dymelor, 500 mg/day. On physical examination he appeared severely ill, and slightly icteric. His abdomen was pathologically enlarged, tympanic but with evidence of ascites.

Chest x-ray revealed air fluid levels beneath the right diaphragm, suggestive of liver abscess or air within the biliary tract. Alkaline phosphatase was 222; LDH, 237; total bilirubin, 2.6; glucose, 223; hemoglobin, 9.4; and hematocrit, 29.1. SBC was 16,200, with differential 91 polys, 5 lymphocytes, 1 stab, and 3 monos. Urinalysis was 1 plus albumin.

Two days following admission, an exploratory laparotomy under endotracheal anesthesia aspirated 500 cc of pus from the right upper lobe of the liver. An operative cholangiogram showed normal anatomy. Culture of the aspirant returned coliform sensitive to Chloromycetin, tetracycline, and Garamycin, with no growth of anaerobes.

The wound was irrigated, and four large drains and a sump drain were inserted. The patient was discharged on the 13th hospital day and readmitted

five weeks later for residual pyogenic abscess resulting from the closure of the drain sinus. Spontaneous drainage at the right upper anterior lateral wall of the abdomen subsequently relieved the pain.

This case is significant in that scintiscan and sonogram were not performed and the large, subphrenic abscess suggested by chest x-ray was not confirmed during surgical exploration. The hepatic abscess was disclosed during surgery through palpation of the dome of the liver.

Case Three: A 44-year-old white female was hospitalized with complaints of increasing epigastric and substernal pain developing during the previous 24 hours. Upon admission, she reported good health prior to the present problem, no vomiting, a slightly elevated temperature (37.9 C) and an intolerance for fatty foods. Physical examination revealed moderate to severe pain on palpation of the epigastrium.

SMAC was normal; WBC was 19.500 with 84% polys, 10 stabs, and 5 lymphocytes. Bilirubin was 1.4 mg; urinalysis was normal. Oral cholecystogram failed to visualize the gallbladder.

When her temperature began to spike to 38.9 C and upper right quadrant pain became severe, she underwent an emergency cholecystectomy. Surgery revealed an acutely diseased and gangrenous gallbladder with pus leaking from the liver. Culture revealed streptococcus sensitive to Erythromycin and Chloromycetin.

The patient was discharged two weeks after admission, with a final diagnosis of acute cholecystitis with cholelithiasis and empyema of the gallblad-

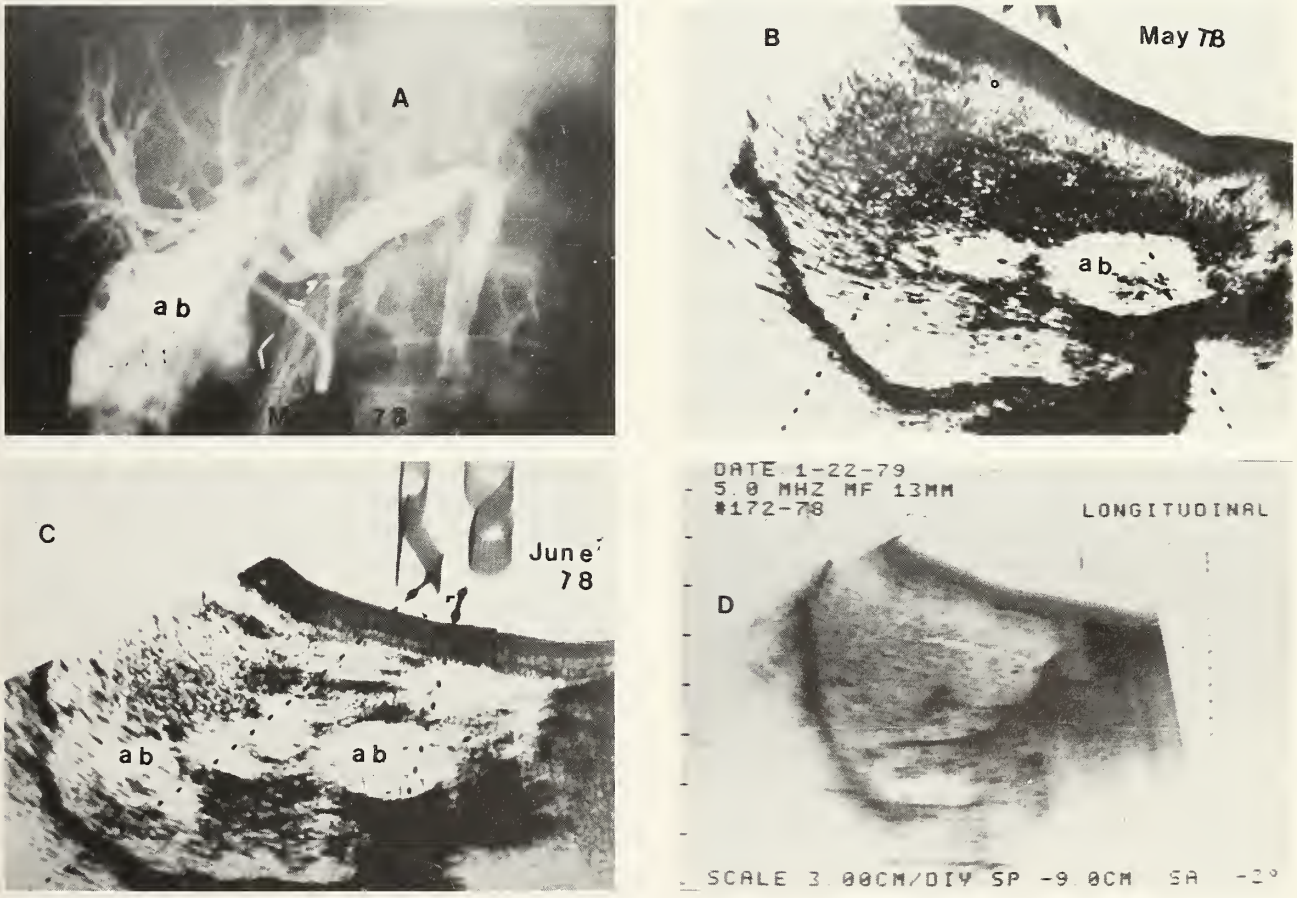


Figure 2. A. A T-tube cholangiogram showing the liver abscess connected with the biliary ducts and a long intraluminal defect in the common duct representing debris. B & C. Follow-up longitudinal ultrasound scan of the hepatic abscess demonstrates increased size and number before drainage of the abscess. D.* Six month follow-up, completely healed hepatic abscess.

*The ultrasound equipment updated to a digital system unit.

der with direct leakage into the liver with small contiguous intrahepatic abscess.

Scintiscan and sonogram were not included in the diagnostic work-up.

Case Four: A 54-year-old white male was hospitalized with chills and fever of several days duration. Tentative diagnosis was either cholecystitis or infected aortic graft. He had been hospitalized two years earlier for an aortic aneurysm and replacement graft and was in the process of recovering when the present complaints began. In addition to chills and fever, there was upper abdominal discomfort.

Heavy antibiotic therapy was begun but symptoms did not remit. After five days, surgery revealed a ruptured gallbladder and cholecystectomy was performed. Operating room cholangiogram revealed biliary ducts connected to a liver abscess. A common duct irrigation was performed, and the right hepatic space was drained. Postoperatively, the chills and fever persisted. Cholangiogram and sonogram revealed multiple liver abscesses. The patient slowly improved and was discharged four weeks after admission with a T-tube in place.

He was readmitted five weeks later with chills and fever, and two weeks later was taken to surgery for drainage of the abscesses. Again the postoperative period was difficult with discharge five weeks after admission. He was again readmitted six months later with a suspected residual abscess which could not be confirmed by ultrasound evaluation *(Figure 2).*

Case Five: A 28-year-old white male was admitted with high fever (40 C) and significant weight loss. His appetite had been fair and he had experienced intermittent nausea and vomiting. The fever was unresponsive to antibiotic therapy; intravenous pyelogram and gallbladder examination appeared normal. WBC was 20,000, and hemoglobin was 10.7 gm. Blood pressure was 129-80; pulse was 80 and regular. The liver was palpable although slightly tender. Alkaline phosphatase was 410, iron 19 mcg, but the rest of the SMAC was within normal limits. WBC differential was 73% polys, 2 stabs, 25 lymphs and overall count of 10.7. Hemoglobin was 11.3; hematocrit, 35.9; platelet count, 409,000; urinalysis was unremarkable. Serum protein electrophoresis suggested liver disease with acute phase of inflammatory reaction. Scintiscan showed a large defect in the right lobe of the liver with indication of multiple small defects throughout, suggesting multiple cysts or abscesses. Sonogram demonstrated a 9 cm sonolucent mass in the right lobe of the liver *(Figure 3).* Chest x-ray was negative, as were barium enema and sigmoidoscopy. Cytomegalovirus results were 1:16.

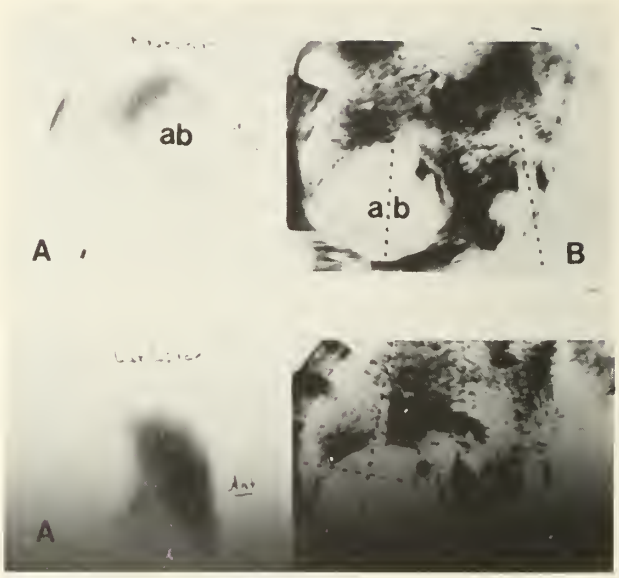


Figure 3. A. Lateral and posterior views of the liver scan showing a large well defined defect in the posterior aspect of the right lobe. B. Transverse sonogram demonstrates about 9 x 9 x 9cm sonolucent with several echoes within it; represents a hepatic abscess.

Two weeks after admission, a laparotomy was performed under general anesthesia and no additional lesions were observed. A secondary incision was made posteriorly, and 500 cc of purulent material was drained from the right lobe of liver.

Cultures were negative and the postoperative course was uneventful. Despite negative culture, the patient was treated with Chloromycetin, 500 mg orally/six hours, gradually reduced to 150 mg. He was discharged three weeks after admission, afebrile but with a persistent alkaline phosphatase of 256.

Summary

Hepatic abscess represents a fairly uncommon but life-threatening condition which can arise from a number of sources. While the infection is usually of either amebic or bacterial origin, the abscess can result from a number of different sources.

Treatment is through surgical drainage and antibiotic support, with success largely contingent upon early diagnosis. Diagnosis can be obfuscated by the rather generalized signs of sepsis in the absence of a primary illness with hepatic abscess as a common sequela.

(Continued on page 362)

Survival

Bypass for Three Vessel Coronary Artery Disease

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NATURAL HISTORY survival studies of patients with coronary artery occlusive disease have shown that the risk factor relates to the number of coronary arteries obstructed. Obstruction of all three coronary arteries has been shown to result in a significantly higher mortality than does more focal (single or two vessel) involvement.[1, 2] If coronary artery bypass favorably affects survival of patients with coronary artery occlusive disease, such an effect should be most readily apparent in this high risk group. The present report attempts to determine whether or not coronary artery bypass has a favorable effect on the five year survival of such patients.

During a six-year period, more than 1300 cases were studied to determine the effect of coronary artery bypass on survival rate of patients with coronary artery occlusive disease. It is apparent that patients undergoing this procedure have experienced an improved five year survival rate. This rate is near the survival rate for a control population of matched age and sex.

Clinical Materials and Methods

During the period between January 1971 and December 1976, 1,326 patients underwent 1,349 isolated coronary artery bypass (CAB) procedures for three vessel coronary artery disease at St. Luke's Hospital. To be included in this group, the patient must have had coronary artery occlusive disease obstructing at least 50 per cent of the luminal diameter of the circumflex coronary artery, anterior descending coronary artery, and right coronary artery. Lesions obstructing the left main coronary artery (in 210 instances) were equated to obstructive disease of both the circumflex and anterior descending coronary arteries. Obstructions of a major distal coronary artery branch (for instance, the posterior descending branch of the right coronary artery or the marginal branch of the circumflex coronary artery) resulted in designating the parent artery as obstructed only if the obstructed branch originally supplied the major distribution of the distal arterial tree. Similar obstructions in only minor branch(es) of one of the three primary coronary arteries did not result in designation of the parent artery as obstructed unless a CAB was performed on the obstructed branch.

No patients were excluded from the study because of preoperative clinical status. Included were patients undergoing emergency operation for preinfarctional angina, evolving or recent myocardial infarction, and in three instances, patients who could not be resuscitated from refractory cardiac arrest. These patients were transferred to the operating room, and were subjected to thoracotomy and institution of cardiopulmonary bypass while undergoing mechanical ventilation and cardiac massage. Patients who underwent CAB in combination with another cardiac or great vessel procedure — such as valve replacement, ventricular aneurysmectomy, resection of an ascending aortic aneurysm — were not included. In summary, this report represents our total experience with isolated CAB for three vessel disease during the six-year period ending December 1976.

Operative death was arbitrarily defined as death occurring within thirty days of operation. The hospital records of patients experiencing an operative death were reviewed and detailed summaries of their courses were made.

Late survival status was determined by office follow-up visits, reports from referring physicians, or telephone interviews with the patients. All patients were eligible for at least a two-year follow-up. Current follow-up data (total follow-up of 5,259.5 patient years) were obtained in all but six patients during the one-year interval ending October 1979. The angina class (based on The New York Heart Association classification) was determined for each

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TABLE I
OPERATIVE EXPERIENCE WITH THREE VESSEL DISEASE

Number of Bypasses	*Operations Performed*	*Operative Deaths*
One	148	3 (2.0%)
Two	611	9 (1.5%)
Three	517	3 (0.6%)
Four or five	73	0
	1,349	15 (1.1%)

interval of the follow-up period. The linearized annual occurrence rates of certain non-fatal events, including acute myocardial infarction and the performance of repeat CAB procedures, were calculated for the first five postoperative years. The cause of late death was determined in each instance. Five year survival of all patients operated upon was calculated by the Life Table Method.[4, 5] The expected five-year survival rate of the United States white population, matched for age and sex, was calculated based on 1970 census data.

Results

The number of operations and the number of coronary artery bypasses performed per operation are shown in *Table I*. Since all patients had significant obstruction involving all three coronary arteries, the number of bypasses performed was determined primarily by the distribution of more distal occlusive lesions, the status of the distal patent arteries, and the presence of left ventricular scar in the region supplied by the arteries. In general, the performance of a smaller number of bypasses implied a less favorable combination of these variables.

The overall operative (30 day) mortality rate was 1.1 per cent. There were 15 operative deaths. The pertinent clinical findings and courses of these 15 patients are shown in *Table II*. Four of the operative deaths occurred in patients beyond the seventh decade of life. Of the nonsurvivors, seven had experienced a previous (as recent as 14 days preoperatively in one patient) myocardial infarction, and two addi-

TABLE II
OPERATIVE DEATHS

Case	*Age-Sex*	*Previous AMI*	*Pre-Op Status**	*LVEF† (%)*	*Bypasses Constructed*	*Cause of Death**	*Time of Death‡*
1	46 M	Yes (14d)	U-III	79	3	AMI	14 d (PD)
2	55 M	No	U-III	70	2	Hemorrhage	3 d
3	71 M	No	U-III	—	2	AMI	1 d
4	71 M	No	U-IV	62	2	LCOS	OR
5	66 M	Yes	U-III	30	1	Sepsis	22 d
6	65 M	Yes	PIA	—	2	AMI	22 d (PD)
7	57 M	Yes	U-IV	—	2	AMI	1 d
8	44 M	No	AMI (ASYS)	—	2	LCOS	OR
9	51 M	Yes	U-IV	35	2	AMI	3 d
10	68 M	No	U-IV	74	1	Aortic Dissection	18 d (PD)
11	72 M	Yes	U-IV	34	2	LCOS	2 d
12	66M	No	U-IV	70	2	PE	14 d
13	54 M	No	S-III	79	3	CVA	29 d (PD)
14	46 F	No	AMI (ASYS)	46	1	LCOS	OR
15	70 M	Yes	S-III	33	3	LCOS	OR

*KEY: U = Unstable or changing angina during 3 mo prior to operation
PIA = Preinfarction angina (continued class IV under treatment in hospital)
AMI = Acute myocardial infarction
S = Stable angina for ≥ 3 mo prior to operation
ASYS = Refractory cardiac arrest
III & IV = Class of preoperative angina (NYHA)
LCOS = Low cardiac output syndrome
PE = Pulmonary embolus
CVA = Cerebrovascular accident

†Left ventricular ejection fraction.

‡Postoperative date of death in operating room (OR), hospital, or postdischarge (PD).

TABLE III
SURVIVAL OF PATIENTS AFTER CAB FOR THREE VESSEL DISEASE

Interval After Operation	*Present At Start*	*Withdrawn Alive*	*Deaths*	*Interval Survival**	*Cumulative Survival† (%)*
0-1 mo	1326	0	15	.9887	98.9 ± 0.6
1-6 mo	1311	2	16	.9878	97.7 ± 0.8
6-12 mo	1293	1	9	.9930	97.0 ± 0.9
1-2 yr	1283	1	16	.9875	95.8 ± 1.1
2-3 yr	1266	199	26	.9777	93.7 ± 1.4
3-4 yr	1041	262	17	.9813	91.9 ± 1.6
4-5 yr	762	251	22	.9654	88.7 ± 2.0

$$*1 - \frac{\text{Deaths}}{\text{Patients at start} - \frac{\text{Patients withdrawn}}{2}}$$

†Survival with 95% confidence (± twice the standard error) limits.

tional patients were infarctional with refractory cardiac arrest at the time of operation. Four operative deaths occurred in patients with severely depressed (LVEF <40%) left ventricular function. Four of the operative deaths occurred after the patients were discharged from the hospital, apparently progressing satisfactorily.

There have been 87 non-fatal acute myocardial infarctions diagnosed during follow-up after discharge from the hospital. This represents a linearized occurrence rate of 1.7 per cent/year. Also, 29 patients have undergone a secondary CAB procedure resulting in an occurrence rate of 0.6 per cent/year of follow-up.

While approximately 75 per cent of all patients were rendered angina free by the operation, there has been a progressive recurrence of angina in many patients. Five years postoperatively 54.4 per cent of all patients are angina free and 73.5 per cent have none or only mild (Class II) angina.

The late survival rate can be surmised from the follow-up data shown in *Table III*. The five year survival rate is 88.7 per cent. It is apparent that the relatively large number of patients followed permits establishment of a high confidence level for the calculated survival rate throughout the first five postoperative years. The expected survival rate of a matched (for age and sex) United States population at five years is 89.9 per cent. It is apparent that the observed five year survival rate is very close to that expected for a matched general population. The causes of the 106 late (>30 days) death are tabulated in *Table IV*. It is apparent that the majority (57%) of late deaths were cardiac in nature.

TABLE IV
LATE DEATHS BY CAUSE

Cause of Death	*Patients*	
Cardiac		
Sudden unexpected death*	32*	60
Acute myocardial infarction	21	
Congestive heart failure	7	
Vascular		
Cerebrovascular accident	12	18
Ruptured aortic aneurysm	3	
Aortic thrombosis	1	
Aortic dissection	1	
Pulmonary embolus	1	
Neoplasia		
Lung carcinoma	8	15
Kidney carcinoma	2	
Brain tumor	2	
Rectal carcinoma	1	
Carcinomatosis	2	
Other		
Pancreatitis	2	13
Suicide	2	
Miscellany†	9	

*Documented ventricular tachyarrhythmias in two patients.
†One patient each due to drowning, intestinal obstruction, dysplastic anemia, amyotrophic lateral sclerosis, cirrhosis, UGI bleeding, pneumonia, sepsis, and emphysema.

TABLE V*
ANNUAL MORTALITY RATES (%) WITH MEDICAL TREATMENT

Reported Series	*One Vessel*	*Two Vessels*	*Three Vessels*
Bruschke *et al.* (1963-1965)	2.9	7.6	10.8
Oberman *et al.* (1965-1970)	2.3	14.5	16.0
Moberg *et al.* (before 1972)	2.9	6.3	10.1
Lichtlen and Moccetti (before 1972)	3.6	6.7	12.6
Slagle *et al.* (before 1972)	4.0	15.4	20.0
Webster *et al.* (1960-1965)	3.3	6.6	10.0
Kouchoukos *et al.* (1970-1974)	1.0	7.5	14.0
Burggraf and Parker (1964-1970)	1.6	7.0	9.0
Lichtlen	2.8	3.9	9.0
Averages	2.7%	8.4%	12.3%

*From Bloomer and Ellestad.[2]

Discussion

A number of studies of the natural history of coronary artery disease, based on angiographically defined pathology, were initated in the early 1960s. A recent review by Bloomer and Ellestad documents the findings of such studies reported during the late 1960s and early 1970s.[2] From the data in these reports they were able to calculate an annual mortality rate based on number of coronary arteries obstructed. These results are shown in *Table V*. In patients with three vessel disease, the mean annual mortality rate was 12.3 per cent. The patients operated upon at St. Luke's Hospital and reported herein have experienced an annual mortality rate of only 2.3 per cent. Admittedly the survival data of the natural history (or medically treated) series are from a different era and many of those patients were not candidates for coronary artery bypass. Therefore, direct comparison between these and the present series of patients must be tempered. However, it is apparent that patients undergoing coronary artery bypass have experienced an improved survival rate and these differences are so striking that the conclusion seems undeniable that coronary artery bypass does (for at least up to five years) improve survival rate. Moreover, it is apparent that the survival rate of the present series of patients is similar to that of the general population when matched for sex and age. There has not been any report of medically treated patients with three vessel coronary artery disease, regardless of how they were selected for therapy, with a five year survival rate approaching that of a matched general population.[7]

It is sobering to note that there is a continuing incidence of certain significant cardiac events (recurrence of angina, acute myocardial infarction, the necessity for repeat coronary artery bypass procedure, and death) which occur in patients even though they have undergone myocardial revascularization. All operative deaths plus late cardiac deaths constituted the majority (62%) of the observed postoperative deaths. These findings point to the palliative nature of myocardial revascularization and illustrate the progressive nature of coronary atherosclerosis.

Conclusions

From our experience and data reported by others, it must be concluded that coronary artery bypass improves the survival of patients with triple vessel coronary artery occlusive disease. How much this procedure improves survival will be answered only with continued follow-up.

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CORRECTION

In the June issue of the *Journal* there was an error in the Current Comment series article, "Evaluation of Anemia." In Table IV on page 298, the Reticulocyte Index heading on the right should have the arrow pointing downward.

The *Journal* regrets this error.

Cimetidine

Treatment of Upper Gastrointestinal Hemorrhage

GEORGE L. MONTO, M.D.,* *and* NIRANDON WONGSURAWAT, M.D.,† *Topeka*

CIMETIDINE, a potent H_2 receptor antagonist, was introduced in 1978 for the suppression of gastric acid secretion. Early symposia have suggested its usefulness in a number of conditions including the management of chronic duodenal ulcer[1] and the treatment of hemorrhage from such ulcers.[2] H_2 antagonists have been considered in treatment of erosive gastritis in the seriously ill patient[3] and for bleeding associated with liver failure.[4] A recent study failed to demonstrate its effectiveness in controlling hemorrhage in four surgical cases with peptic ulceration.[5]

To clarify the usefulness of cimetidine in the treatment of acute upper gastrointestinal (UGI) hemorrhage other than variceal bleeding and hemorrhagic disorders, the following study was conducted. The hypothesis that cimetidine decreases the duration of gastrointestinal hemorrhage in ulcero-erosive disease was tested by studying patients with gastrointestinal hemorrhage from various sites, before and after its introduction to this hospital in May 1978. Student's "T" test was used to compare the difference in duration of hemorrhage in patients treated with parenteral cimetidine and in a control group. Attending physicians were not influenced regarding methods of treatment.

A 24-month study of 31 consecutive cases was utilized to determine the effectiveness of the H_2 antagonist cimetidine (Tagamet) in treatment of upper gastrointestinal hemorrhage. Fifteen patients were treated with intravenous cimetidine and 16 were treated by conventional methods. It appears that cimetidine treatment did not significantly decrease the duration of bleeding in ulcero-erosive gastrointestinal hemorrhage in this population.

Patients and Methods

More than sixty consecutive cases of gastrointestinal hemorrhage were screened during a 24-month period. Charts were reviewed and all patients were seen by one of the authors. Cases were considered for study when good documentation of the bleeding site was confirmed, and in those patients without evidence of liver disease or bleeding tendency on routine automated screening tests of SGOT, bilirubin, alkaline phosphatase, and prothrombin time. Bleeding was confirmed by medical personnel on admission of the patient to the hospital. The day of admission was considered the first day of bleeding. Diagnostic studies included emergency gastroscopy in one half of the cases. Such study was done in patients with rapid bleeding from an unknown site. In more stable patients without imminent prospect of surgery a careful UGI series was performed as the main diagnostic test. UGI x-ray studies were performed on all patients. Two or more criteria described later were used to determine the time of cessation of bleeding. Cases without such documentation of bleeding site or accurate determination of duration were excluded, reducing the study to a total of 31 patients.

Fifteen patients were treated with conventional therapy plus cimetidine 399 mg intravenously/six hours. Sixteen controls were treated conventionally with antacids and nagogastric suction. Thus, 16 cases were similarly treated except for the addition of cimetidine to the regimen for one group. Four patients were assigned randomly to one or the other group. Treatment by various attending physicians was carried out in all other patients which included cimetidine in almost all cases after May 1978. Patients who required more than five units of blood were transferred to the Surgical Service to be evaluated for laparotomy. In some instances the patients ceased bleeding prior to surgery. Surgery was required to bring about cessation of bleeding in two cases in each group. Cessation of bleeding was determined endoscopically in a small number of cases.

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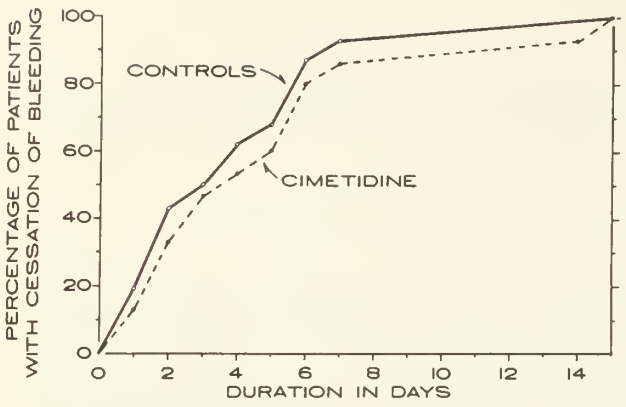


Figure 1. Cumulative percentage of patients with days of bleeding in cimetidine treated cases (dashed lines) and controls (solid line). Similar curves and duration are observed.

Stability of hemoglobin levels for 48 hours, clearing of blood from nasogastric tube drainage, and absence of gross and occult blood in the stool were used as additional criteria. Difference in the duration of bleeding in the two groups was determined by Student's "T" test.

Results

Nine duodenal ulcers, four instances of gastritis, one of esophagitis, and one stress ulcer were the causes of bleeding in the cimetidine treated subjects. In the control patients, duodenal ulcer was found in ten cases, gastritis in four, esophagitis in one, and Mallory-Weiss syndrome in one. The two groups were comparable as to bleeding sites.

Figure 1 is a cumulative tabulation of the number of days of bleeding in the two groups of patients. The cimetidine treated patients appear to have a slightly slower resolution of bleeding, but this is not statistically significant. All patients ceased bleeding by the fifteenth day. One patient in each group died after cessation of bleeding; both of these patients were elderly and one in the cimetidine treated group had undergone surgery.

Average age in the treatment group was 45, and in controls 62. This would tend to favor the younger patient with presumably greater tissue and vascular reserves. Apparently no such effect was shown. Neither group had any apparent complication of treatment. Mean duration was 5.1 days $\pm$ 4.27 in the cimetidine treated group. Mean duration of bleeding in the control group was 4.0 $\pm$ 3.49 days ($p > 0.95$). Thus, no significant reduction in duration of bleeding was found; in fact, bleeding may have been prolonged. Four patients began bleeding while being treated with oral cimetidine.

Comment

Despite the fact that no study has demonstrated conclusively the usefulness of cimetidine for UGI bleeding, it is generally available as an intravenous substance and is used extensively for this purpose in this community. A study of duration of bleeding from acid peptic disease was done to determine whether this agent given parenterally brought about cessation of bleeding more quickly than conventional methods of treatment. Although it clearly decreases gastric acid output and is useful in chronic duodenal ulcer treatment,[2] the role of cimetidine as a therapeutic agent in gastrointestinal hemorrhage is unclear. No conclusive beneficial effect in UGI hemorrhage was demonstrated in our study. This concurs with the results of an earlier smaller series study from England.[5]

On the whole, short term treatment with cimetidine has been found to be safe. No complications from short-term treatment with cimetidine were found in this study. Elevations in serum creatinine and transaminase concentrations have been reported, although the meaning of these changes is unknown.[6] In the patients in this study, no such elevations were noted during cimetidine treatment. Patients with elevated SGOT, bilirubin, and alkaline phosphatase were not, however, included in this study because of the possibility of pre-existing liver disease with its effect on hemostasis.

Alternative treatment methods were not specifically studied in this series because of the wide variety of treatments used here including nasogastric suction with or without iced saline lavage, antacid therapy ranging from hourly to four times/day, and occasionally milk drip. Frequent antacid therapy was most frequently used in this series with nasogastric suction being the second most popular method of treatment. Antacids in very large doses were shown to accelerate ulcer healing in a large cooperative study, although symptomatic relief was not demonstrated.[7] The effectiveness of antacids in gastrointestinal hemorrhage has not been studied in any large group although they continue to be advocated.[8] A recent study of cimetidine *vs* antacids in the prevention of stress ulceration indicated superiority of antacid drip in the critically ill patient.[9] It is the opinion of the authors that intracellular buffering may be reduced and that there may be inadequate reduction of intralumenal buffering in cimetidine treated patients.

(Continued on page 356)

Acoustic Tumors

Surgical Results

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C. KEITH WHITTAKER, M.D., *Kansas City, Missouri*

Twenty patients with acoustic tumors underwent surgery during the period of October 1976 through April 1980. The operative results of these consecutive cases reveal that there were no fatalities, total tumor removal was obtained in all cases, and complete absence of facial function occurred in only two patients.

Acoustic tumors most commonly present with hearing loss and tinnitus. Every patient whose complaints are unilateral hearing loss and tinnitus must be thoroughly evaluated.

Evaluation of patients with symptoms of an acoustic tumor has changed over the years. Impedance audiometry has replaced older audiometric tests such as Bekesy, SISI, and ABLB. Brainstem electric response audiometry shows promise as the most valuable audiometric test. Tomograms of the internal auditory canal are commonly performed in most hospitals. Electronystagmography is routinely used in vestibular evaluation.

The most revolutionary advance in radiology has been the computed cranial tomogram. This has not replaced the posterior fossa pantopaque cisternogram but is a complementary study. Acoustic tumors less than 1.5 cm in size are routinely missed on computed tomograms and usually will not be found unless cisternography is performed. In fact, computed cranial tomography may have set back the early diagnosis of acoustic tumors[1] because the physician assumes the patient does not have a tumor if the computerized scan is negative. As will be pointed out in this paper, this is not the case.

Sponsored by the Midwest Ear Research Institute, 2945 Wyandotte Street, Kansas City, MO 64108.

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Case Material

From October 1976 through April 1980, 20 patients with acoustic tumors underwent surgery. All of these patients presented with hearing loss and balance disturbance. These cases are presented in consecutive order in *Tables I* and *II*.

TABLE I
POSTOPERATIVE VII NERVE FUNCTION WITH NON-DISPLACEMENT OF IV VENTRICLE IN 20 PATIENTS

Case	*Age*	*Pantopaque*	*C-T*	*VII Function*	*Follow-up*
1	52	+	–	+ + +	39 mo
3	58	+	–	+ + +	35 mo
8	46	+	–	+ +	24 mo
9	52	+	2.0 cm	+	21 mo
10	65	+	–	+ + +	19 mo
12	63	+	–	+ + +	14 mo
13	35	+	–	+ +	13 mo
17	63	not done	1.6 cm	+ + +	1 mo
18	29	not done	0.9 cm	+ + +	1 mo
20	35	not done	0.45 cm	+ + +	2 wk

TABLE II
POSTOPERATIVE VII NERVE FUNCTION WITH DISPLACEMENT OF IV VENTRICLE IN 20 PATIENTS

Case	*Age*	*Tumor Size*	*VII Function*	*Follow-up*
2	36	1.8 cm	+ + +	38 mo
*4	56	3.5 cm	0	31 mo
5	68	3.2 cm	+ +	30 mo
6	57	3.0 cm	+	29 mo
7	51	3.2 cm	+ +	28 mo
11	21	2.5 cm	+ + +	15 mo
14	55	2.0 cm	+ + +	13 mo
15	54	1.7 cm	+	4 mo
*16	26	3.8 cm	+ +	2 mo
19	64	2.5 cm	0	1 mo

*Preoperative hydrocephalus.

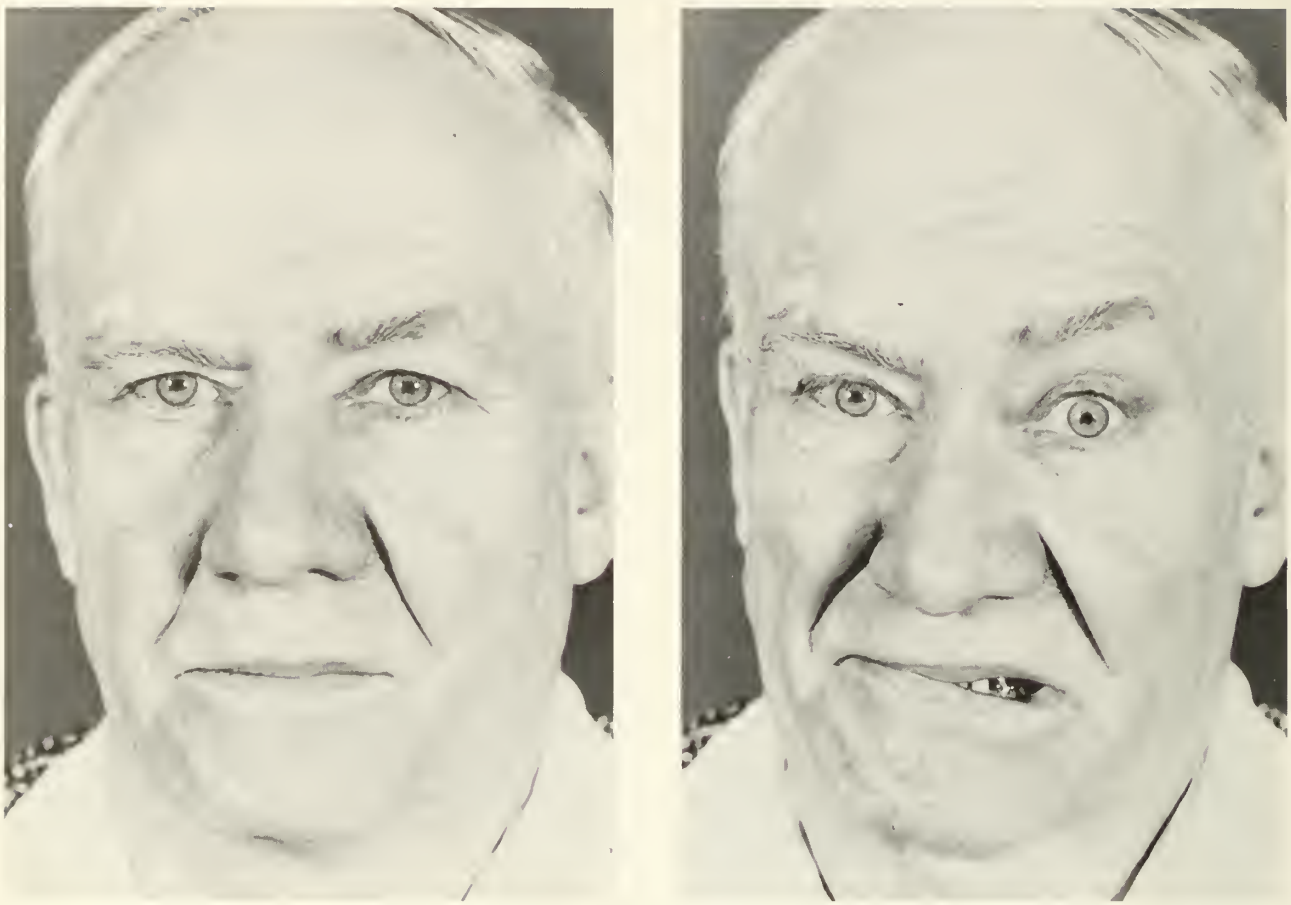


Figure 1. Facial function 18 months post-operatively in sixth patient who underwent rerouting of facial nerve and VII-VII anastomosis at brainstem. A. In repose. B. With attempted full facial movement.

Fourteen of the 20 patients had positive C-T scans. In 12 cases where the scan was positive, a pantopaque study was not performed. Two of the cases with positive C-T scans had a pantopaque study previously performed, one 11 months previously, and in the second case, following the C-T scan. Both of these patients were referred following diagnostic studies. Tumor size ranged from 1.0 to 2.0 cm in the six patients with negative C-T scans.

Tumor size in this series ranged from 1.0-4.5 cm. Determination of tumor size was based upon configuration of the tumor on C-T scan, cisternogram, and correlation with the surgical tumor bed following removal.

Immediate facial function (in recovery room) and the current (follow-up) status of facial function are compared in *Tables I* and *II*. A + + + indicates normal facial function with no minimal defect. A + + designation represents less than normal facial function, complete eye closure, with or without some synkinesis, and normal appearance in repose. A + designation indicates some movement of facial musculature, but not acceptable as a good result, and incomplete eye closure although normal facial appearance in repose.

Tables I and *II* show the 20 cases divided into two groups depending upon displacement or non-displacement of the IVth ventricle. This division is correlated with facial nerve function.

The fourth patient in this series with a 3.5 cm tumor had no facial function post-operatively and the facial nerve was sacrificed. The sixth patient in the series had the facial nerve rerouted from the temporal bone and anastomosed to its entry zone at the brainstem. The nerve had been splayed out by tumor close to the brainstem and was interrupted in order to insure total tumor removal. He had return of facial function to the lower and midface 15 months post-operatively. Forehead function is minimal, but his face is normal in repose *(Figure 1)*.

TABLE III
VARIATION IN SURGICAL PROCEDURE

Case	*Tumor Size C-T*	*Variation in Procedure*
4	3.5 cm	Secondary suboccipital procedure for total removal at 3 months
6	3.0 cm	Rerouted VII nerve from mastoid to VII nerve stump at brain stem
8	C-T NEG	Suboccipital, transmeatal approach to preserve hearing; unsuccessful
11	2.5 cm	Planned one-stage, combined suboccipital, translabyrinthine
16	3.8 cm	Middle fossa transtentorial, suboccipital, translabyrinthine

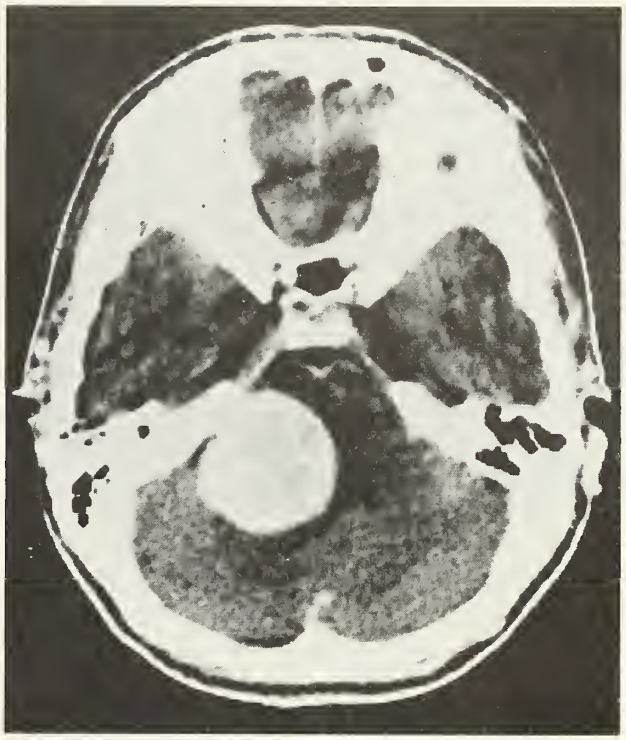


Figure 2. C-T scan 3.8 cm right acoustic tumor.

Although 16 of the 20 patients were operated exclusively through the translabyrinthine approach, there were variations in the surgical procedure in Cases 4, 8, 11, and 16 *(Table III)*. In the fourth patient, approximately 50 per cent of the tumor was gutted with the House-Urban dissector through the translabyrinthine approach. The remainder of the tumor was removed three months later through a suboccipital approach. The eighth patient was operated through a suboccipital transmeatal approach in an attempt to preserve hearing. This patient had a 2 cm tumor, and audiogram indicated good speech discrimination. Although we were able to obtain total tumor removal and near normal facial function, we were not able to preserve hearing. A planned one-stage combined suboccipital and translabyrinthine approach was used in the 11th patient. The 16th patient had a 3.8 cm measured tumor by C-T scan with hydrocephalus and papilledema *(Figure 2)*. By any standard, this was a large tumor. A planned one-stage combined middle fossa transtentorial, suboccipital, translabyrinthine approach was utilized in this patient with total tumor removal and immediate near normal facial function (+ + result).

Follow-up ranged from 2 weeks to 3.5 years. There were no fatalities and total tumor removal was obtained in all cases.

Discussion

The goal of acoustic tumor surgery is total tumor removal with preservation of normal facial function. Preservation of hearing would be ideal. Preservation of hearing is possible approximately one-half of the time when tumors are small and limited to the internal auditory canal.[2] There is debate regarding the preservation of cochlear function through the suboccipital transmeatal approach for tumors 2 cm in size or less.[3-5] Glasscock has proposed extending the middle fossa approach for preservation of hearing to tumors up to 1.5-2.0 cm by ligating the superior petrosal sinus and opening the tentorium to obtain a view of the posterior fossa.[6]

The future will bring more attempts at preservation of hearing. However, preservation of facial nerve function remains difficult in all cases and should remain of prime importance. Ideally, both should be preserved.

The advantage to the translabyrinthine approach is positive identification of the facial nerve at a point not involved by tumor where the nerve exits from the internal auditory canal.[7] This allows tumor removal with the nerve in view at all times. This approach offers rapid access to the surgical field in case of postoperative hemorrhage. The tumor bed may be reached by quickly opening the postauricular incision, removing the fat that is used to close the defect and allowing the blood clot or hemorrhage to decompress itself. This can be done in the recovery room or intensive care unit and the patient then taken to the operating room for definitive control of hemorrhage. The translabyrinthine approach allows the surgeon to develop the capsule of the tumor, keeping the arach-



Figure 3. C-T scan of eleventh patient with 2.5 cm right acoustic tumor.

noid between the surgeon and the major blood vessels. Utilizing the House-Urban rotary dissector, the center of the tumor can be decompressed and the capsule of the tumor developed. The major disadvantage of the translabyrinthine approach is the lack, at times, of posterior surgical exposure.

The dura may be incised behind the sigmoid sinus and the posterior aspect of the tumor dissected from the cerebellum.[1] This combined approach was utilized in the 11th case. This patient had a 2.5 cm right acoustic tumor *(Figure 3)*. The translabyrinthine exposure was accomplished identifying Bill's bar, the facial nerve, and superior vestibular nerve. The dura was not yet opened. A plate of bone was then removed from behind the sigmoid sinus and the dura opened through a suboccipital approach. Nerves IX, X, XI were identified and the tumor gutted from posteriorly toward the brainstem. When the dura was opened through the translabyrinthine approach, the tumor had been gutted and reduced to approximately 1-1.5 cm in size. This facilitated easier removal of the tumor from the facial nerve and brainstem. Facial function was normal at the completion of the operative procedure.

Another advantage to the translabyrinthine approach — well demonstrated by case six — is the ability to reroute the facial nerve from the temporal bone and anastomose it at the brainstem.[8] The facial nerve is usually splayed out from the tumor and dissection is difficult at the porous and medial to the internal auditory canal. The distal segment can be rerouted and sutured to the stump of the facial nerve at the brainstem. This is impossible through the suboccipital, transmeatal approach.

Tumor size is often difficult to estimate. The surgeon often over estimates the size of the tumor at the operating table. This has led to inaccurate description of size. Many classifications have been proposed for judging tumor size. All tumors should be accurately recorded in centimeters by use of the cursor on C-T scanners and reported as such. This series utilized C-T scanning; therefore, if the C-T scan was negative and the cisternogram positive, no measurement was given. Tumor size is listed as measured on C-T scan. Measurements given in *Tables I* and *II* are cursored C-T measurements of tumor in the cerebellopontine angle. Improved resolution allowed visualization of a 0.45 cm extension into the angle in case 20.

Complications

In this series of 20 patients with acoustic tumors, there were four complications directly attributable to the surgical procedure *(Table IV)*.

The sixth patient was placed on steroids and the night of the second postoperative day, developed abdominal discomfort and distention. He ruptured a colonic diverticulum and developed peritonitis requiring a diverting colostomy which was subsequently closed. He also developed cardiac arrhythmias. This case points out the value of an internist in the management of these patients.

The second and third complications were in the tenth and eleventh patients who developed a CSF rhinorrhea requiring reclosure of the wound.

The 16th patient developed a minor wound infection. The 19th patient required aspiration of a hema-

TABLE IV
POSTOPERATIVE COMPLICATIONS

Case	*Complication*
6	Ruptured colon diverticulum with peritonitis on POD #3 (on steroids)
10	CSF rhinorrhea requiring secondary closure on POD #12
11	CSF rhinorrhea requiring secondary closure on POD #28
16	Postoperative wound infection
18	Urinary tract infection from Foley catheter
19	Abdominal wound hematoma requiring evacuation

toma from the abdominal wound donor site used for fat closure of the translabyrinthine defect.

None of these patients have had further neurological deficits other than abnormal facial function (as seen in *Tables I* and *II*) and loss of hearing.

We stress a team approach in the care of these patients. Considerable time and effort have been spent through in-service education of hospital personnel. It is safe to say the ruptured diverticulum and peritonitis of the sixth patient could very well have been fatal had it not been for astute diagnosis and management.

We do not use antibiotics in these patients prophylactically. Since the release of cimetidine in the United States, we use this with pre-operative steroids in patients with larger tumors.

Summary

Twenty patients underwent gross total removal of acoustic tumors from October 1976 through April 1980. Mortality rate for this series is zero; total tumor size varied from 1.0-4.5 cm. These cases were approached predominantly through the translabyrinthine exposure with the exception of Cases 4, 8, 11 and 16. Absence of facial function is present in only two patients beyond two-week follow-up. These results compare favorably with other reports *(Table V)*.

TABLE V
SUMMARY OF POSTOPERATIVE RESULTS

Gross total removal	100%
Mortality rate	0%
Facial paralysis	*10%

*19th patient; 1 mo follow-up included VII anatomically intact.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Iron Overload

SEAN R. LYNCH, M.D.,* *Kansas City, Kansas*

The body of a normal adult male contains approximately 3 gm of metabolically active iron. In addition there is an iron store which ensures the availability of iron for essential physiological processes. The size of this reserve is determined by the balance between the amount of available dietary iron eaten and individual iron requirements. Normally regulation of the rate of iron absorption prevents stores from exceeding approximately 2 gm; however, they may increase beyond these limits as the result of excessive absorption, repeated blood transfusions, or parenteral iron administration. This situation, irrespective of pathogenesis, is referred to as siderosis, hemosiderosis or preferably, iron overload. Progressive enlargement of iron stores eventually results in tissue damage. The term hemochromatosis describes conditions leading to severe iron overload (body iron stores of greater than 15 gm) together with cirrhosis of the liver or other tissue damage resulting from the presence of iron.

Table I lists the causes of iron overload. Idiopathic hemochromatosis and hemochromatosis secondary to ineffective erythropoiesis, particularly in association with thalassemia major, are the most common.

Idiopathic Hemochromatosis

Idiopathic hemochromatosis (IHC) may be considered the prototype for all of these conditions. It results from the absorption of excessive quantities of iron from a normal diet, but the mechanism responsible for the defective control of iron absorption remains obscure. Most patients develop clinical symptoms during middle age when the body iron content is between 15 and 40 gm. Current evidence suggests this is the result of an initial absorption rate of 3-5 mg of iron/day. Absorption decreases progressively as stores enlarge, and eventually equilibrium is reached when absorption and excretion rates are approximately equal. The body tolerates considerable increases in iron stores; however, tissue damage ulti-

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TABLE I
CLASSIFICATION OF IRON OVERLOAD

Primary	Increased absorption	Idiopathic hemochromatosis
Secondary	Parenteral iron	Post transfusional Excessive parenteral iron therapy
	Increased absorption ± parenteral iron	Iron-loading anemias (ineffective erythropoiesis) Thalassemia major Sideroblastic anemia
	Increased absorption	Dietary iron overload Associated with liver disease Alcoholic cirrhosis Following portacaval anastomosis Associated with porphyria cutanea tarda

TABLE II
CLINICAL FEATURES OF IDIOPATHIC HEMOCHROMATOSIS

Symptoms	*Signs*
Weakness and malaise	Hepatomegaly
Diabetes	Pigmentation
Pigmentation	Loss of body hair
Weight loss	Testicular atrophy
Loss of libido	Spider nevi
Abdominal pain	Splenomegaly
Dyspnea	Arthritis
Peripheral neuritis	Ascites
	Cardiac failure
	Jaundice

mately occurs and the patient then enters the clinical phase of the disease.

Clinical Features: Although many different organs may be affected in IHC, the most characteristic abnormalities are found in the liver, skin, certain endocrine organs, the joints, and the heart *(Table II)*. The liver is usually the first organ affected, and hepatomegaly is found in almost all symptomatic patients. Unless the individual drinks excessive amounts of alcohol, deterioration of liver function tends to be more gradual than in Laennec's cirrhosis. Carcinoma — usually in the form of hepatoma — is an important late complication seen in approximately 15 per cent of patients.

Skin hyperpigmentation, like hepatomegaly, is almost always present although it may be mild. It is usually generalized but most prominent in the sun exposed areas, genital regions, and scars. The oral mucosa also is pigmented in about 10-15 per cent of patients. The most commonly encountered endocrine abnormalities are diabetes — present in 30-60 per cent of patients — and hypogonadism. Loss of libido, impotence, or amenorrhea are almost inevitable in younger subjects. Other forms of endocrinopathy are less frequently encountered. Although only recently recognized, arthropathy has been reported to occur in 25-91 per cent of cases; clinical syndromes include chondrocalcinosis and a slowly progressive degenerative arthropathy initially involving the metacarpophalangeal and interphalangeal joints of the hands. The latter condition bears a superficial resemblance to rheumatoid arthritis. The most striking features of heart disease in IHC are congestive failure and cardiac arrhythmias. These are often presenting features in young people and may cause death within a year unless therapy is instituted.

Diagnosis: The diagnosis of IHC depends upon the demonstration of three features: (1) Increased total body iron; (2) Absence of other known causes of iron overload; and (3) A family history of iron storage disease. When clinical findings suggest the diagnosis of hemochromatosis, an estimate of the size of body iron stores should be obtained on the basis of investigations listed in *Table III*. If values for serum iron concentration, percentage saturation of transferrin and serum ferritin level are normal, further testing is not necessary and clinically manifest IHC can be excluded. When one or more of the measurements is above the normal range, initial screening should be followed by determination of urinary iron excretion in response to desferrioxamine (24-hour urinary iron excretion following the intramuscular administration of 500 mg desferrioxamine).

A definitive diagnosis of IHC usually depends upon liver biopsy for routine histological assess-

TABLE III
INVESTIGATION OF IRON OVERLOAD

Investigation	*Normal Range*	*Idiopathic Hemochromatosis*
Serum iron (μg/dl)	65-165	>170
Percentage transferrin saturation (%)	20-50	>50 (usually >80)
Serum ferritin (μg/l)	12-250	>300 (usually >1000)
Urinary iron* (mg)	<2	5-20 (usually >8)
Stainable hepatic iron (grade 0-4)	0-1	3-4 (Parenchymal distribution)
Hepatic iron concentration (μg/gm dry wt)	300-2250	>4500

*24-hr excretion after desferrioxamine 500 mg IM.

ment, histochemical estimation of tissue iron, and if possible, chemical measurement of hepatic iron concentration. Liver biopsy should be undertaken only if screening tests, including urinary iron excretion after desferrioxamine, suggest the presence of iron overload. Predominantly parenchymal localization of hemosiderin permits IHC to be distinguished from other varieties of iron overload. Micronodular cirrhosis is almost always seen, although lesser degrees of fibrosis have been observed in young patients. The final and most accurate assessment of the amount of excess iron present in the body at the time of diagnosis is obtained retrospectively by calculating the quantity of iron removed during treatment by repeated phlebotomy.

Screening of Relatives: Once the diagnosis of IHC has been established, all first-degree relatives over the age of ten years should be screened for the presence of excessive iron stores. Recent studies of families with IHC have indicated that it is usually inherited as an autosomal recessive trait and that at least one IHC susceptibility gene is located on chromosome 6 in close proximity to the HLA A locus. While the specific HLA haplotype associated with the IHC susceptibility gene will differ in different families, HLA A_3 and either HLA B_7 or HLA B_{14} are encountered more commonly in patients with IHC than in the general population. It is possible to use HLA typing to identify relatives of patients with IHC who are at risk for developing iron overload. However, despite the potential value of HLA typing, the combined measurement *(Table III)* of serum iron concentration, percentage saturation of transferrin, and serum ferritin level remains the best screening regimen for IHC. If the initial screening is negative, the evaluation should be repeated at yearly intervals.

Treatment: The consequences of organ damage are managed along conventional lines, and excess iron is eliminated by instituting a phlebotomy program. Careful assessment of the heart is important before therapy is initiated. In the presence of increased cardiac irritability, phlebotomy should be carried out slowly under careful supervision and continuous infusions of desferrioxamine may be advisable during the initial period to protect the myocardium from free iron mobilized by the procedure. Once excess iron has been removed and the serum iron and serum ferritin are in the normal or iron deficient range, phlebotomy need only be done two to six times/year. Iron stores can be monitored conveniently by measuring the serum ferritin once a year. The level should be held below 200 μg/l.

There is good evidence that phlebotomy improves longevity. The five-year survival rate in one study was increased from 33 to 89 per cent. Damage to certain body tissues is arrested; improvement in cardiac and hepatic function is usually most noticeable. In contrast gonadal function, diabetes, and arthropathy appear to be little altered by therapy in most patients. Unfortunately, treatment of some of the more rapidly progressive consequences of chronic iron toxicity and extension of life have lead to an increase in the incidence of neoplastic complications. Hepatocellular carcinoma now accounts for approximately 30 per cent of all deaths in IHC. This finding emphasizes the importance of diagnosing and treating IHC in the precirrhotic phase, although it is not yet known whether removal of iron at this stage will reduce the risk of hepatocellular carcinoma.

Secondary Iron Overload

Secondary iron overload occurs either as the result of repeated blood transfusions or because of excessive iron absorption. In patients with hypoplastic anemia, the main source of excess iron is blood transfusion. Those who have anemia in association with a hyperplastic bone marrow — usually secondary to ineffective erythropoiesis — accumulate iron both as the result of blood transfusion and increased absorption. Excessive iron absorption may also result from increased dietary iron content, although significant iron overload with gross deposition of iron in the liver is rare in the absence of other disorders. One notable exception occurs in the South African black where the incidence of iron overload at necropsy in adult males may be as high as 70 per cent.

Iron overload has been reported in association with liver disease, following portacaval anastomosis, and in association with porphyria cutanea tarda. Finally a slight to moderate increase in stainable hepatic iron is common in alcoholic cirrhosis; liver iron concentration is usually less than twice the upper limit of normal, and total iron stores rarely exceed 3 gm. In a small proportion of patients with alcoholic cirrhosis, the hepatic iron concentration is markedly elevated. Some of these patients have identifiable contributing factors such as ineffective erythropoiesis or portal-systemic shunting, and recent studies suggest that the majority of the remaining patients are in fact suffering from IHC.

Patients with secondary iron overload may develop all the features of IHC. Hepatic parenchymal iron deposition is most prominent when the source of

the excessive iron is the gastrointestinal tract. Nevertheless, only a small proportion of the South African black population with iron overload develops parenchymal iron damage and organ failure similar to IHC. In patients with hypoplastic bone marrows where the major source is blood transfusion, the primary localization of the excess iron is reticuloendothelial. Although redistribution occurs with time, there is initially less impact on the parenchymal cells of the liver.

Treatment: In patients with normal bone marrow function, phlebotomy remains the most suitable therapeutic option; however, in many patients with secondary iron overload this is not possible. Interest in the iron chelating agent, desferrioxamine (DFO), has been reawakened recently by studies demonstrating that significant quantities of iron may be removed from the body by using large doses of DFO administered over prolonged periods of time (1-4 gm of DFO administered during a 12-24 hour period will achieve a negative iron balance in most patients, suffering from thalassemia major). The practical application of this therapy has been made possible by the demonstration that subcutaneous administration is almost as effective as intravenous infusion and the development of a portable pump to infuse DFO subcutaneously during a 12-24 hour period. Children can receive the chelation therapy with sedation during a 12 hour period at night and continue normal activities during the day.

This dose of DFO induces the urinary excretion of 1-4 mg iron/kg/day. In addition, there is an increase in excretion of iron through the gastrointestinal tract. Removal of 1-2 gm iron/month is adequate to keep the average patient receiving 4-6 units of blood/month in negative iron balance. The use of DFO in combination with the maintenance of a normal or near-normal hemoglobin level in children with thalassemia major represents a very important advance in the management of this condition.

Self Assessment Questions

1. Idiopathic hemochromatosis in the clinically manifest phase of the disease can be excluded by the presence of a normal serum iron, percentage saturation of transferrin, and serum ferritin level. (T or F)
2. Loss of libido, impotence, and amenorrhea are uncommonly encountered in idiopathic hemochromatosis. (T or F)
3. Treatment of symptomatic idiopathic hemochromatosis by phlebotomy substantially improves life expectancy. (T or F)
4. The arthropathy associated with idiopathic hemochromatosis disappears following phlebotomy therapy (T or F)
5. Treatment with adequate doses of desferrioxamine may lead to negative iron balance in patients with secondary iron overload. (T or F)

(Answers on page 360)

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The President's Message



We all seem to be prejudiced to varying degrees. It is evident in our humor (ethnic jokes, political jokes, woman drivers, etc.) and it certainly is reflected in the popularity of Archie Bunker, in whom we all see a bit of ourselves to laugh at. But of all the areas in which we may find a reason to be prejudiced — religion, color, sex, social status, professional status, politics — the most curious must surely be our apparently universal, though oft times subtle, negative attitude toward the aged. They are slow in the midst of a busy world. They are forgetful in spite of critical medication schedules. They are talkative but we have no time to listen. They have insurance papers to fill out which they cannot comprehend. They have illnesses for which we have no cure.

Life expectancy at birth is now 73 years. It is indeed peculiar in our youth oriented society that we seem to be so biased against the one state in life nearly all of us will attain — advanced years, aging, old age — call it what you will. If you have all of the answers but nobody asks you the questions, you may already be there.

One area of importance to our senior citizens is long term health care, particularly in nursing homes. This year we will have a committee looking into nursing home care from the patients' point of view, as well as the doctors' and owners' points of view. I hope we can give attention to an important area of health care that the Kansas Medical Society has tended to neglect perhaps as a cumulative subconscious prejudice toward old folks. But as Pogo says of his enemy, someday "we is they."

Fraternally,

President



Editorial COMMENT

Gender Role

It has been noted that the cause of feminism has been well served this year by the winning of the Kentucky Derby and the near winning of the Belmont Stakes by a filly. And as every woman knows, an occasional touch of adversity as demonstrated at Pimlico can do wonders for the cause at times. The more militant members of the movement might consider this a frivolous reference, considering their long and arduous struggle for a fair share of the sunlight. Before they turn us off, then, let us say that this is leading up to the fact that one stated intent of our scarcely-broken-in president, Phil Godwin — to bring female physicians into a more active and visible function in the Kansas Medical Society — is off and running.

We are not privy to the details of Dr. Godwin's thoughts and certainly have no intention of usurping his plan but it seems appropriate to offer a little support to the effort by some observations drawn from the records of the Society.

The first female physician to gain membership in the Kansas Medical Society was one Francena R. Porter of Lawrence. She was proposed for membership (the proposer unknown but, of necessity, male) at the annual meeting in Leavenworth in 1872, and while the official record is sketchy, the Secretary exhibits a certain perplexity in the handling of the unprecedented situation since the applicant is referred to as "Miss Francena R. Porter, M.D." (At least he didn't call her "Ms.") As was customary, her application was referred to the Board of Censors (again obviously all male). Since the Board's report was rendered later at the same session, we can visualize a hurried putting-together of heads to ponder the matter. The Board cautiously returned a favorable verdict contingent upon evidence that the lady's alma mater, Woman's Medical College of Philadelphia, was recognized by the "regular profession." Apparently, someone was able to offer this assurance as she was subsequently elected to membership by a vote of 20 yeas and 5 nays whereupon parliamentary gallantry asserted itself, and the vote was amended to be unanimously favorable. The lady was thereupon formally introduced and signed the Constitution (the Society's, that is) and medical history, female division, Kansas chapter, was made. It seems doubtful, however, that the Secretary fully recovered as he subsequently entered her in the minutes as "Doctoress Porter."

It would be good to report that the doctoress and the Society rose to glorious heights together through their mutual efforts, but the scanty record fails us. Dr. Porter was appointed to the Special Committee on Obstetrics in 1873. She is not recorded as a member of the committee after that, but it was customary to change the membership each year so it was, in itself, no reflection on the lady. She attended the annual meeting in Lawrence in 1874, and then is lost to organizational records.

There was apparently a gap in time before other female physicians arrived on the scene, but Dr. Porter must have cleared the way since subsequent female applicants were accorded only the standard scrutiny. The count of female members in those years is a little uncertain as some used initials only, and whether this was a conscious expression of unisex intent or for typographical convenience isn't clear. Then there is some confusion in the relationship between membership in the Society and the "holding of a certificate." The Society, in the days before the Medical Practices Act established a state board of licensure, assumed the obligation of holding examinations and issuing certificates of medical proficiency. In 1880, Julia B. Brown, Olathe; Sarah C. Hall, Fort Scott; Deborah K. Longshore, Topeka; Minerva Lewis, Lawrence; E. B. (sic) Slosson, Sabetha; and Nancy Welles, Topeka, were both certified and KMS members. A Mrs. R. S. Tenney, of Independence, passed the examination, but her medical qualifications otherwise are unknown and she doesn't seem to have joined the club if she had a medical degree. Augusta Conry, Florence, in 1882;

Ruth M. Wood, Leavenworth, in 1884; and Adeline Metcalf, Topeka, in 1888, were added. In 1883, Dr. Longshore was elected second vice-president of the Society, a fact which, given the general attitude of the times, says more for the lady than the elimination of sexual prejudice. There must have been some coming and going as the roster for 1902 lists five identifiable females, for a 2.2 per cent portion of the 238 total membership.

At this point, it should be said that any reference to figures is little more than casual, not only because we disbelieve most of those we read (or the way they are used at least) or that the record is uncertain as already noted, but mainly because the numbers are too small to support anything more than suggestions of effect. Even now, the medical body is fragmented by active physicians, academics who combine teaching or research with varying amounts and types of active practice, public health physicians, administrators, and so on. This situation is related to the increasing number of physicians and the development of diverse responsibilities as the population has grown and social and cultural changes have occurred. It has been apparent in the female group as well with, in some instances, the inevitable requirement of sharing their professional duties with their domestic demands. We can note that there are presently 126 female members of the Kansas Medical Society in its total membership of 2650 — about 4.75 per cent.

One can say that 126 is a respectable figure or even point out that the percentage is double that of the turn of the century, but neither concept is particularly satisfying. What is important is what they are doing and where they are doing it. It cannot be questioned that female physicians have made significant advances in status, recognition, production, and contribution nor can it be questioned that the climate must be made suitable for more which is undoubtedly the thrust of Dr. Godwin's planning. And we can note that their advances have accompanied the progress of women in all areas of endeavor, but we can say that women have achieved more in the practice of medicine in this period of transition than they have in other disciplines. This is not only because we believe (with admitted professional chauvinism) that the medical function is superior to the others but (and partly in consequence) women who have pursued medical careers have had to overcome deeper and more rigorous prejudices — outside the profession as well as within — than others. They have had to meet a combination of physical, intellectual, cultural and emotional demands of singular character, and we suspect they have contributed more to the groundswell of women's rights activities than they have received. And they owe their male colleagues very little in the process.

Out of this, however, comes one figure of significance. The current proportion of females in our medical school classes is running 20-25 per cent, and there can be no doubt of the validity of this gain, its importance as a gain, and its viability. The obvious result will be a significant increase in the number of women among practicing physicians. The quality has long been established and now the quantity can begin to exert increasing effect. There will be an evening of sexual representation and influence in all branches of medicine. Their predecessors mentioned above were largely locked into obstetrics or gynecology or pediatrics — at least non-surgical and non-male procedures. If it appears to have been a condition compatible with their own choice, it was a choice dictated by social attitudes — and abetted by their male compatriots.

But at the same time, the process is accentuating the infrequency with which female members of the profession are involved in the functions of organized medicine. Some of them would undoubtedly attribute this to a continued state of prejudice on the part of the males, but to whatever extent this may be true, it is much less so than in even the recent past. It may be, too, that the struggle for equality produces a degree of independence (or requires it to begin with) which carries over and sensitizes the female physician against what she sees as a still-male bastion.

This, then, is our interpretation of Dr. Godwin's intent — to identify the female physicians who are not members of the KMS and encourage their membership; to utilize those who are members more effectively by creating an awareness among them that the opportunities are there; and to hasten the day when a common functional identity will apply to the physicians in the state without regard to sex. This is not to say that female physicians should not retain their specific capacity for service which relates to that peculiar neuroendocrine complex which defines them as feminine — we trust that will never be lost. But it does mean that the conditions are right for a genuine equitability of effect in the affairs of the Society. As important as any encouragement from within will be the willingness on their part to participate in the process. — *D.E.G.*

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Cimetidine

(Continued from page 342)

Another treatment to accelerate ulcer healing may be carbinoxalone which is available in England. This compound may strengthen mucosal resistance by promoting synthesis of gastric mucosal glycoprotein, thereby protecting mucosal surfaces from ulceration.[10, 11] Laser beam coagulation of bleeding ulcers is effective although its clear superiority has not been demonstrated.[8] These methods have not been fully evaluated and it would appear that no single method of treating gastrointestinal hemorrhage has yet been established as clearly superior to another. Our study shows no effect of the recently introduced substance cimetidine on duration of bleeding compared to conventional methods; larger prospective studies of various agents are indicated to ascertain if any single treatment method is most effective.

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.: A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction, changes in EEG patterns may appear during and after treatment blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets

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September 26, 1980

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The 388-page volume, prepared by the AMA's Center for Health Services Research and Development, is comprised of statistical information from the AMA Physician Masterfile, the most comprehensive and complete source of physician data in the U.S.

The volume answers such diverse types of questions as how many of the total medical doctor population of 437,486 practice aerospace medicine; how many of the 45,540 women physicians are engaged in surgical specialties; how many doctors practice forensic pathology; and how many doctors live in Montana.

The work also contains data covering licenses issued to physicians by licensing boards, policies of state boards of medical examiners for initial licensure for graduates of U.S. medical schools, trends in the distribution of physicians, concentration of specialties and information on hospitals, population and income.

Information covering the ratio of physicians, excluding those in government (federal), in the U.S. reveals that New England has the highest ratio (238) and the lowest (134) is in the South Central division (Alabama, Kentucky, Tennessee and Mississippi).

The latest U.S. physician ratio to population of 187 per 100,000 is approximately 23 per cent more than the 1971 figure of 152 physicians per 100,000 population.

While 1978 figures are not available to compare the U.S. ratio to European countries, information supplied by the World Health Organization shows that for 1975, the ratio in France is 1 physician for every 650 persons; in Italy the ratio is 1 physician to every 490 persons; in West Germany, it is 1 physician for every 500 persons, in Sweden it is 1 physician for every 580 persons and in Great Britain it is 1 physician for every 760 persons.

In the area of Physician Manpower, the section describes the distribution of 375,811 physicians who are professionally active in the U.S. and possessions. The majority of active physicians, 342,714 or 91.2 per cent, are classified in "patient care." Of the physicians engaged in "patient care," 54,893 are in general practice which includes family practice, 96,940 are in medical specialties, 98,567 are involved in surgical specialties and another 92,314 in "other specialities."

Of the 45,540 women physicians, approximately 6,688 more than in 1976, 11,979 are engaged in medical specialties and another 12,251 are involved in "other specialties."

Of the 56,197 physicians engaged in general practice, 21,611 are engaged full time in family practice medicine.

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The volume in addition includes data on the 20,242 physicians who work for the federal government. Most of the number, 41.3 per cent, are located in the southern region and particularly in the South Atlantic Census Division. A total of 12.6 per cent of the federal physician population is engaged in "administration," while 9.4 per cent is involved in "research."

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Iron Overload

(Continued from page 351)

Answers

1. *True*
2. *False*
3. *True*
4. *False*
5. *True*

Suggested Readings

1. Crosby, W. H.: Hemochromatosis: The unsolved problems. *Semin. Hematol.* 14:135, 1977.

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Brief Summary of Prescribing Information

Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability an insomnia associated with anxiety neuroses and transient situational disturbances, anxie associated with depressive symptoms and as a treatment of symptoms of anxiety if such sym toms are a significant feature of functional or organic disorders, particularly gastrointestinal cardiovascular

Effectiveness in long-term use, i e , more than 4 months, has not been assessed by syste atic clinical studies Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses As with all CN acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and diminished tolerance for alcohol and other CNS depressants

Physical and Psychological Dependence Withdrawal symptoms like those noted with bar turates and alcohol have occurred following abrupt discontinuance of benzodiazepin (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating) Addi tion-prone individuals, e g drug addicts and alcoholics, should be under careful surveillan when on benzodiazepines because of their predisposition to habituation and dependenc Withdrawal symptoms have also been reported following abrupt discontinuance of benzod azepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid ove sedation

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result symptoms like those being treated anxiety, agitation, irritability tension, insomnia and occ sional convulsions

Observe usual precautions with impaired renal or hepatic function

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepa has not been shown of significant benefit in treating gastrointestinal or cardiovascular comp nent

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year 6mg kg day No effect dose was 1 25mg kg day (approximately 6 times the maximum hum therapeutic dose of 10mg day) Effect was reversible only when treatment was withdrawn wit 2 months of first observation Clinical significance is unknown, but use of lorazepam for pr longed periods and in geriatric patients requires caution and frequent monitoring for sympto of upper G I disease

Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia, some have h elevations of LDH As with other benzodiazepines, periodic blood counts and liver function te are recommended during long-term therapy

CLINICALLY SIGNIFICANT DRUG INTERACTIONS Benzodiazepines produce CNS depress effects when administered with such medications as barbiturates or alcohol

CARCINOGENESIS AND MUTAGENESIS No evidence of carcinogenic potential emerged rats during an 18-month study No studies regarding mutagenesis have been performed.

PREGNANCY Reproductive studies were performed in mice, rats, and 2 strains of rabb Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastrosch malformed skull and microphthalmia) were seen in drug-treated rabbits without relationsh dosage Although all these anomalies were not present in the concurrent control group, have been reported to occur randomly in historical controls At 40mg/kg and higher, there evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lo doses Clinical significance of these findings is not known However, increased risk of cong tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam meprobamate) during first trimester of pregnancy has been suggested in several stu Because use of these drugs is rarely a matter of urgency, use of lorazepam during this pe should almost always be avoided Possibility that a woman of child-bearing potential may pregnant at institution of therapy should be considered Advise patients if they become pr nant to communicate with their physician about desirability of discontinuing the drug

In humans blood levels from umbilical cord blood indicate placental transfer of lorazepa and its glucuronide

NURSING MOTHERS It is not known if oral lorazepam is excreted in human milk like oth benzodiazepines As a general rule, nursing should not be undertaken while on a drug sin many drugs are excreted in milk

Adverse Reactions, if they occur, are usually observed at beginning of therapy and gener disappear on continued medication or on decreasing dose In a sample of about 3,500 anxio patients most frequent adverse reaction is sedation (15 9%), followed by dizziness (6 9% weakness (4 2%) and unsteadiness (3 4%) Less frequent are disorientation, depression, na sea change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, e function disturbance various gastrointestinal symptoms and autonomic manifestations. In dence of sedation and unsteadiness increased with age Small decreases in blood press have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind that multiple age may have been taken Manifestations of overdosage include somnolence, confusion and co induce vomiting and/or undertake gastric lavage followed by general supportive care, monit ing of vital signs and close observation Hypotension, though unlikely, usually may be control with Levarterenol Bitartrate Injection U S P Usefulness of dialysis has not been determined

Ativan®(lorazepam) for Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dos gradually when needed, giving higher evening dose before increasi daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosa may vary from 1 to 10mg/day in divided doses. For elderly or debi tated, initially 1-2mg/day; insomnia due to anxiety or transient situ tional stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Are you concerned about long-acting metabolites? Many clinicians, as well as pharmacologists, are beginning to draw attention to this problem (see New England Journal of Medicine, April 5, 1979).

In contrast to some older benzodiazepines, Ativan (lorazepam) does not give rise to long-lasting active metabolites. As with all benzodiazepines, you should follow the usual precautions concerning co-administration with other CNS depressants and warn your patients against operating dangerous machinery and motor vehicles.

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Ativan has a convenient b.i.d. or t.i.d. dosage schedule; it is compatible with a long list of other medications and, of course, it is a highly effective anxiolytic agent, as established in numerous nationwide, double-blind, controlled evaluations in thousands of patients.



See important information on preceding page.

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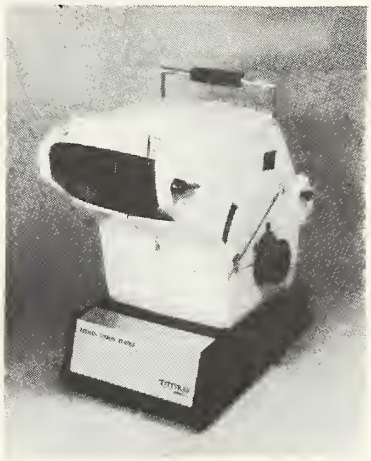
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HOW TO WRITE A MANUSCRIPT?

1. Cultivate an undecipherable handwriting and under no circumstances resort to printing. If you must print, be sure to print all capital letters so typist will not know which words to capitalize. This will help appreciably in confusing the typist and eliminate the possibility of accurate final results.
2. Smooth out your "V"s to resemble "U"s so that whether it is "Value" or "Valve" will be anyone's guess. Don't overlook the fact that the difference between "Altitude" and "Attitude" could make a specification sound like a punch line from the George Gobel show.
3. Check to be sure sentences are grammatically constructed so as to be incomplete. Always leave split infinitives and dangling participles, and use verbs only in case of extreme emergency. Change tense several times throughout the message.
4. When writing numerals several times throughout one draft, be sure that they are written differently each time to eliminate the possibility of detection. Switch from the right to the left hand if you find duplicate numbers are getting to look too much alike.
5. Do not number chronologically when enumerating. It is better to put paragraph one at the bottom of the page and draw arrows. This same rule applies to putting the last paragraph first and drawing arrows to the bottom. This can be especially interesting if arrows extend through several pages.
6. Whenever possible, write long unpunctuated sentences, changing the thought trend several times to achieve complete incomprehensibility. Everyone will then think you are a genius, and in no time at all, you will find yourself being quoted.
7. Do not date material to be typed. This eliminates the possibility of anyone discovering who goofed and makes passing the buck more of a community project.
8. To forestall all possibility of work being completed on time, be sure to copy it at least two weeks late. *Typing skill is developed through panic.*
9. Be sure to use a hard lead pencil so it will be difficult to read.
10. In the interest of saving paper, write the last ten sentences in tiny lettering and squeeze onto the last half inch of the paper. Also, another interesting project is to add some paragraphs to previous pages, but don't make any notation at the spot — wait until about 20 to 40 pages down the way, then write "place this paragraph insert on page 1"; make the insert large enough that it will move forward all the rest of the text, illustrations, and tables. This is guaranteed to cause a few dozen comments about the writer from the typist.
11. Spelling should be fairly close, but try to leave out a letter here and there. When you resubmit for typing over, you can also correct your more serious goofs at the same time to justify the retyping.

This "How to" article, by an anonymous author, appeared in *Scope*, a house organ of Hope Medical Center, Duarte, California. It was reprinted in *Medical Communications* 7(1):ii. It is reprinted here with the permission of both publications.

Hepatic Abscess

(Continued from page 336)

Diagnosis can be greatly facilitated through the use of scintiscan and sonogram techniques. These techniques have additional value in monitoring postoperative changes, response to antibiotic regimens, and recurrence of abscess.

Five cases of hepatic abscess have been used to emphasize the protean nature of the causes and presenting complaints, and to illustrate both the need for and the advantage of consistent use of scintiscan and sonogram techniques in cases of suspected hepatic abscess.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
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Product Information as of September, 1977

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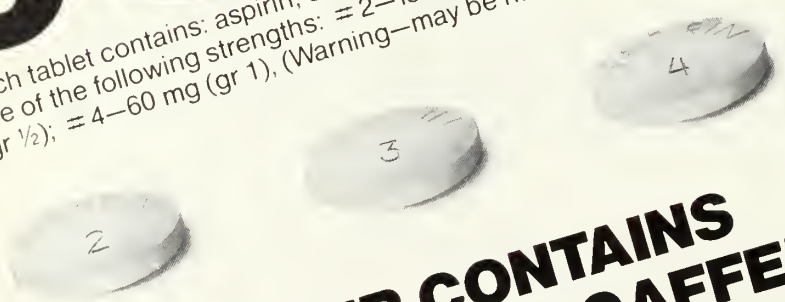
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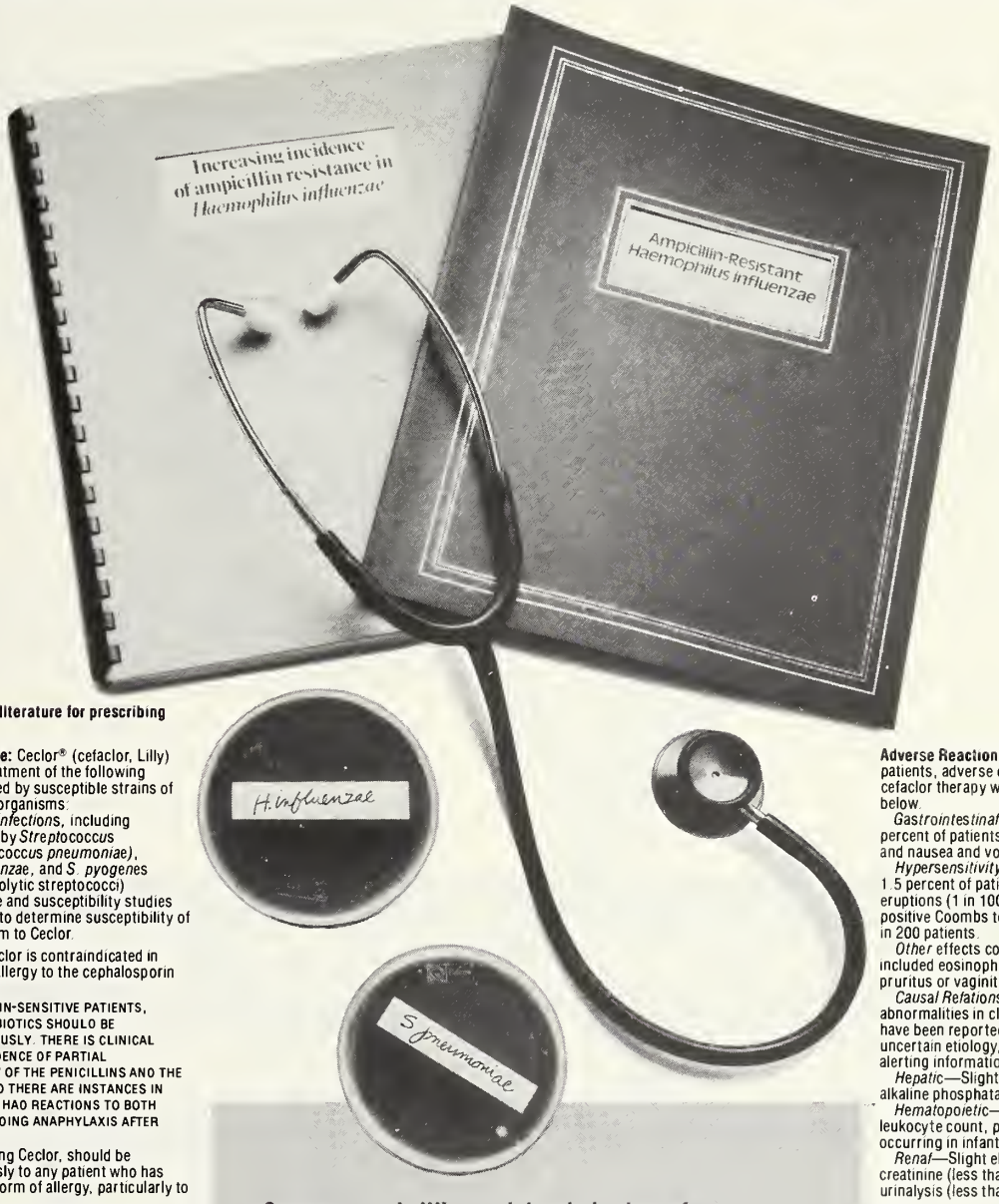
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Brief Summary
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about 2.5 percent of patients and included diarrhea (1 in 70) and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in about 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less than 1 in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

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Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

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*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor® (cefaclor) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

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Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

General guidelines for the prescribing and appropriate use of minor tranquilizers

- Individualize dosage for maximal beneficial effect.
- Prescribe the specific quantity needed until the next checkup period, schedule frequent, periodic reexaminations to monitor results of therapy.
- Establish treatment goals and gradually discontinue medication when these have been met.
- Avoid prescribing for individuals who appear dependency-prone or whose histories indicate the potential for misuse of psychoactive substances, including alcohol.
- Caution patients against engaging in hazardous occupations requiring complete mental alertness such as operating machinery or driving.
- Advise patients against the ingestion of alcoholic beverages while undergoing therapy with minor tranquilizers.
- Counsel patients to follow label directions, keep medication out of children's reach, and dispose of unused or old medication.
- Caution patients against giving medication to others.
- Avoid abrupt cessation of extended therapy by tapering dosage before discontinuing medication.

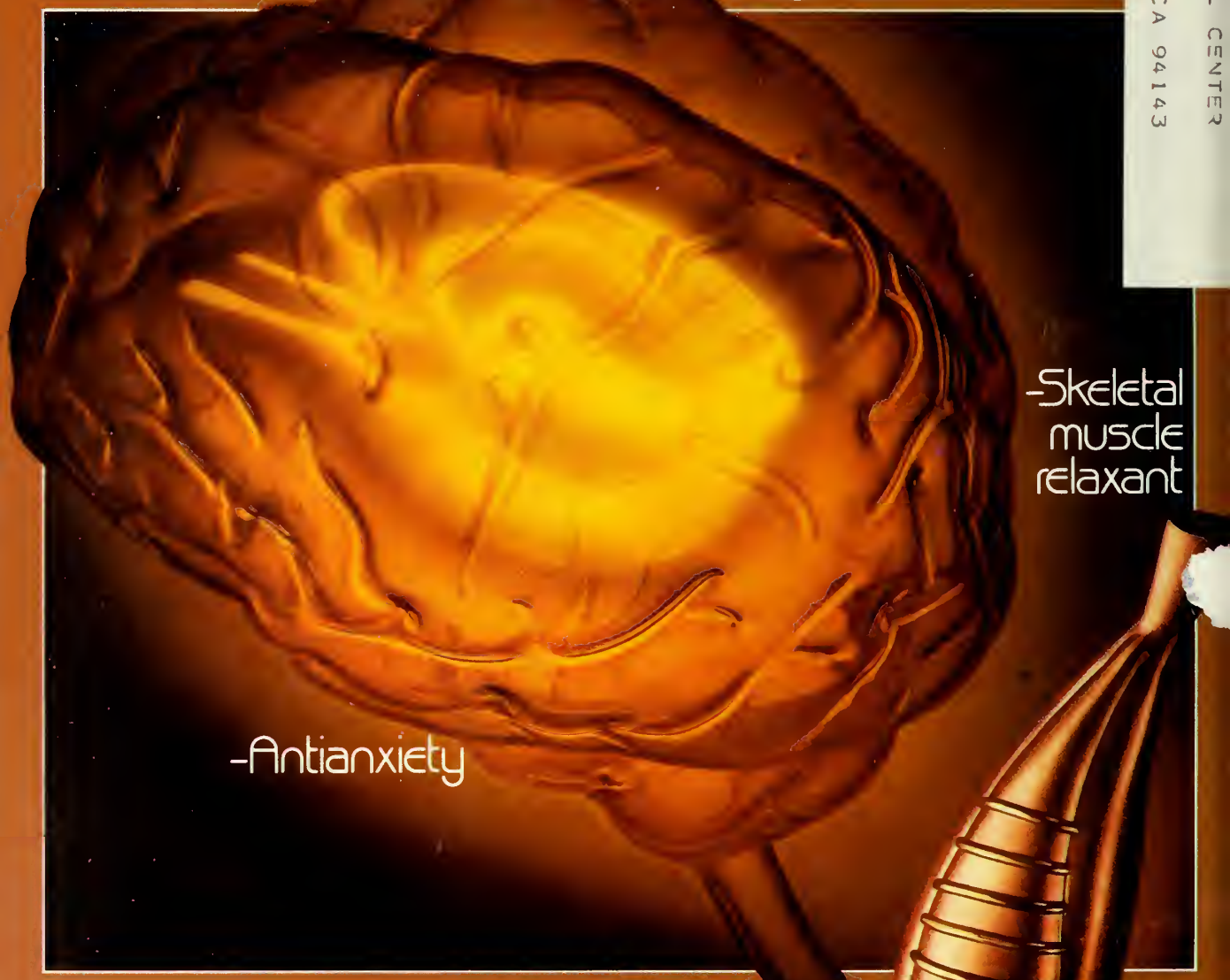
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-Antianxiety

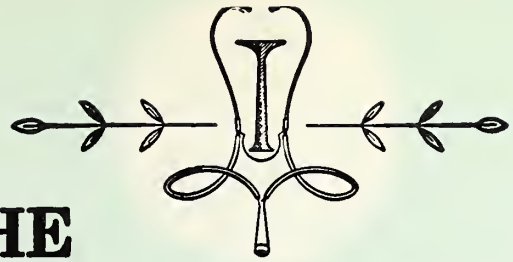
2-mg, 5-mg, 10-mg
• scored tablets

Valium®(V)
diazepam/Roche

Indicated in anxiety and tension states and as an adjunct in the relief of skeletal muscle spasm

Please see summary of product information on preceding page.

ROCHE®



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THE Journal of the Kansas Medical Society

AUGUST
1980

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VOL. LXXXI
NO. VIII

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Volume LXXXI, Number 8 — August 1980

The JOURNAL of the KANSAS MEDICAL SOCIETY

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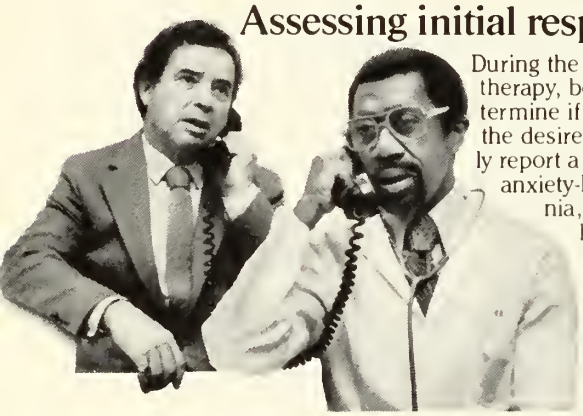
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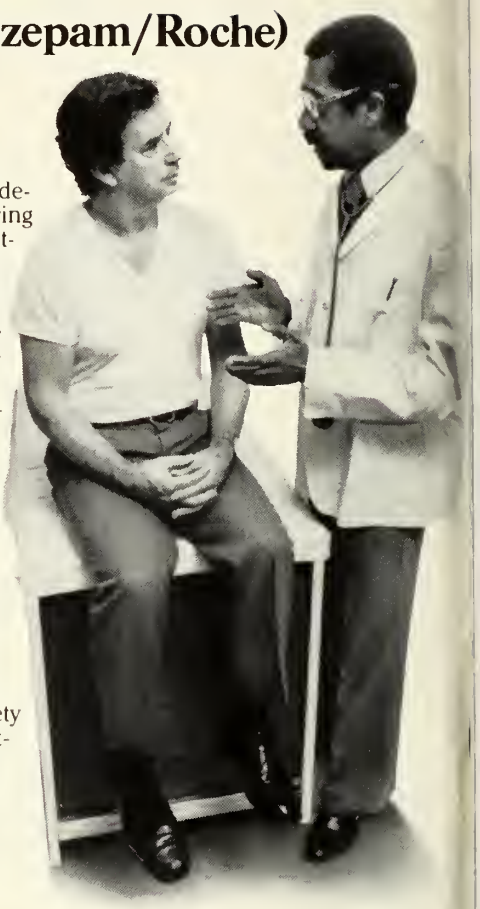
PLEASE NOTE: This roster has been designed for insertion into the *Physician Membership and Practice Guide* (red notebook) currently being distributed to all members.

Monitoring patient response to Valium® (diazepam/Roche)

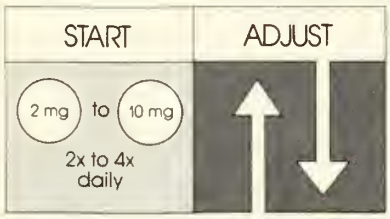
Assessing initial response to therapy



During the first follow-up visit after initiating therapy, both physician and patient should determine if Valium (diazepam/Roche) is having the desired effect. Most patients will promptly report a feeling of relaxation and relief of anxiety-linked symptoms such as insomnia, headaches, palpitations and hyperventilation. You will probably observe that the patient is calmer and more relaxed. If, however, patient response does not measure up to expectations, a reevaluation of the patient's profile with modification of the dosage regimen should be considered.



Making dosage adjustments



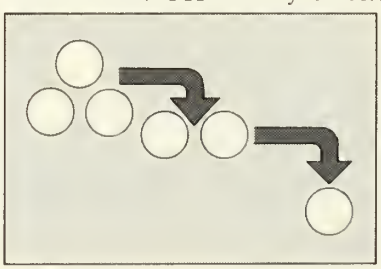
With any psychoactive medication it is good medical practice to initiate therapy at base dosage levels and titrate to the patient's needs. With Valium, experience has shown that 5 mg t.i.d. is usually sufficient although some patients with severe or persistent anxiety may require higher dosages initially. In geriatric or debilitated patients, the recommended dosage is 2 to 2½ mg once or twice daily. When anxiety fluctuates, as is common with most patients, the dosage may be adjusted as needed during the course of therapy; three strengths in scored tablets give you unmatched flexibility and simplicity in individualizing dosage.

Evaluating progress toward therapeutic goals

SET GOALS						
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

At the beginning of therapy it is now common practice for both physician and patient to establish treatment goals and to estimate the amount of time needed to achieve them. Then the patient knows what to expect and when to expect it. Some physicians find that compiling a checklist of presenting symptoms and complaints is useful for assessing the patient's response from visit to visit. In this way, progress toward attainment of the therapeutic goal is reviewed at regular intervals. As patients feel their symptoms abate and begin to develop insight into the sources of their anxiety and psychic tension, the checklist can be expected to dwindle.

Discontinuing pharmacologic intervention



When you decide to discontinue therapy, tapering dosage is good medical practice. Although rarely necessary after short-term treatment with Valium, gradual dosage reduction is advisable for patients who have been on extended therapy. This gradual discontinuance should preclude either recurrence of pretreatment symptoms or development of untoward side effects. Symptoms of withdrawal have almost always been associated with abrupt discontinuance of therapy after higher dosages taken continuously over long periods of time.

2-mg, 5-mg, 10-mg scored tablets

Valium® (IV)
diazepam/Roche

An Important Adjunct to Your Treatment Program for Excessive Anxiety

ROCHE®

See the following page for a summary of product information.

Valium®(diazepam/Roche)℞

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome, convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

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Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

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Tenuate®
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATION Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence*. Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic* Urticaria, rash, ecchymosis, erythema, *Endocrine* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico 00633

Direct Medical Inquiries to
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

Licensor of Merrell®

References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.: A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

Merrell

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Overweight may not always be simple... complications can develop*.

Complicated or not...

Tenuate® Dospan® ℂ IV

(diethylpropion hydrochloride NF)

75 mg. controlled-release tablets



A useful short-term adjunct in an indicated weight loss program.

Overweight patients in certain diagnostic categories often require strict appetite control and a successful program of weight reduction may tend to diminish the incidence or severity of the complications in some patients. Diethylpropion hydrochloride has been reported useful in such patients and while it is not suggested that Tenuate itself in any way reduces the complications of overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. **Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on the opposite page.**

In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness.

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate—it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

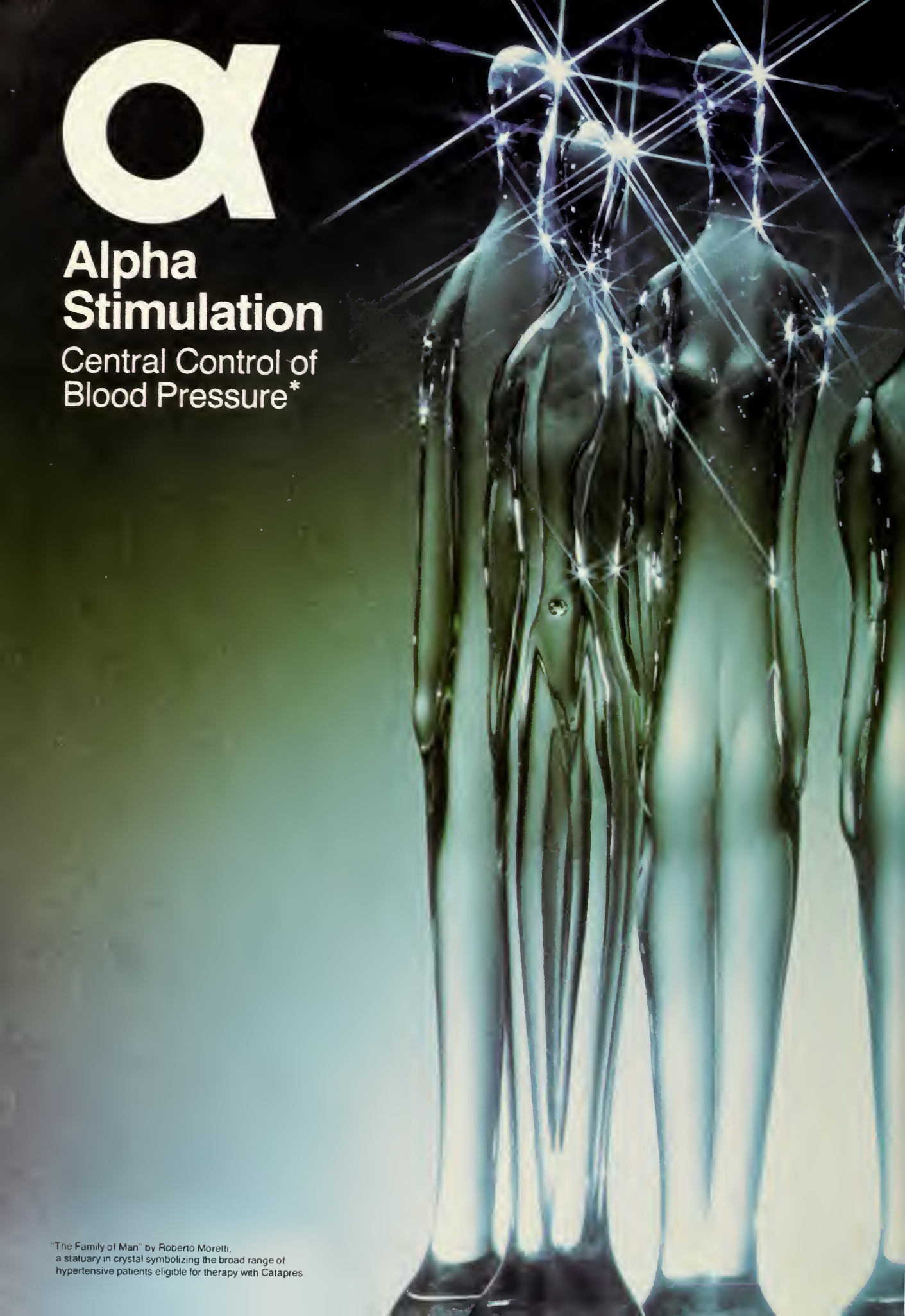
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For prescribing information see opposite page.

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Hyperglycemia	Hyperuricemia
Diabetes mellitus	Gouty arthritis
Bronchial asthma	Sulfonamide hypersensitivity

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work/play—normal hemodynamic responses to exercise maintained.

love—low incidence of impotence and/or loss of libido: 2.8% in 1,923 patients studied.[1]

cardiac output—tends to return to control values during long-term therapy.

blood flow—preserved in kidney.

No Single Advantage Determines Drug Choice.

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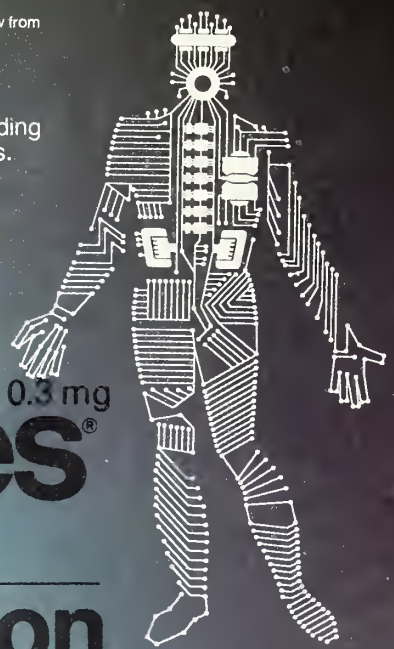
The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

1. Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

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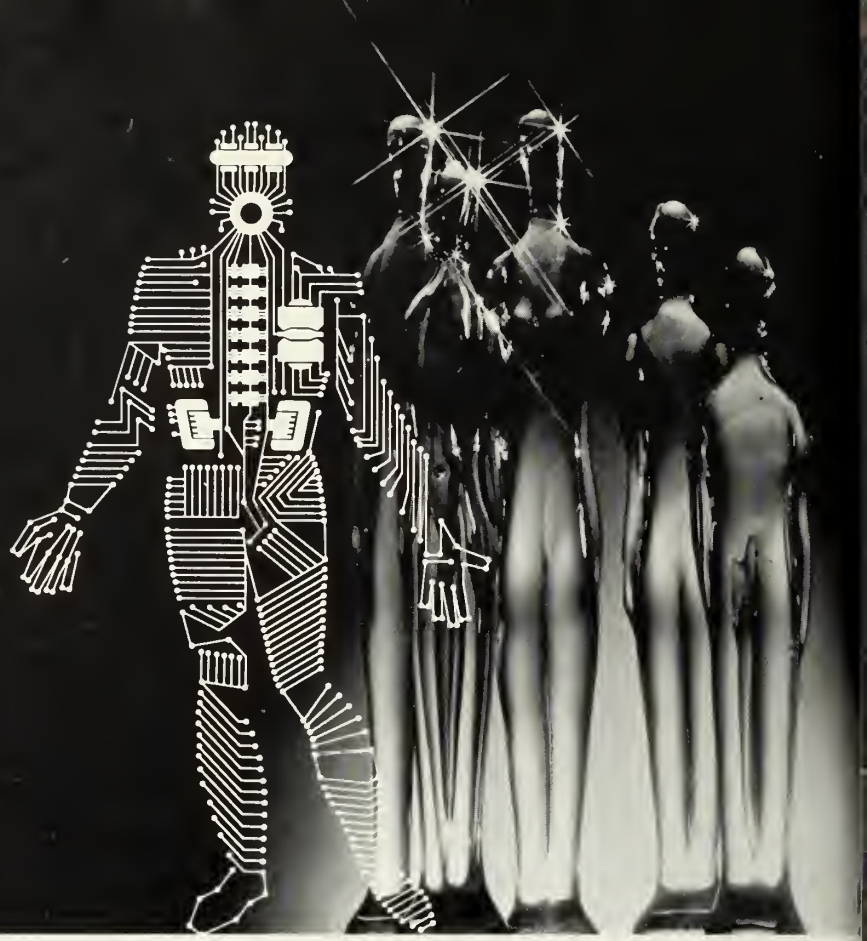


Tablets of 0.1, 0.2, 0.3 mg

Catapres®
(clonidine HCl)
Hypertension

The Alpha Advantage:

It's for all kinds of hypertensives



Tablets of 0.1, 0.2, 0.3 mg

Catapres®
(clonidine HCl)
Hypertension

- No contraindications.
- Effective in all degrees of hypertension. It is mild to moderate in potency.
- Low incidence of depression, impotence, orthostatic hypotension—no fatal hepatotoxicity.
- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
(clonidine hydrochloride)
Tablets of 0.1, 0.2, 0.3 mg

Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase; congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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ANUSOL-HC® SUPPOSITORIES
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ANUSOL-HC® CREAM
Rectal Cream with Hydrocortisone Acetate
Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

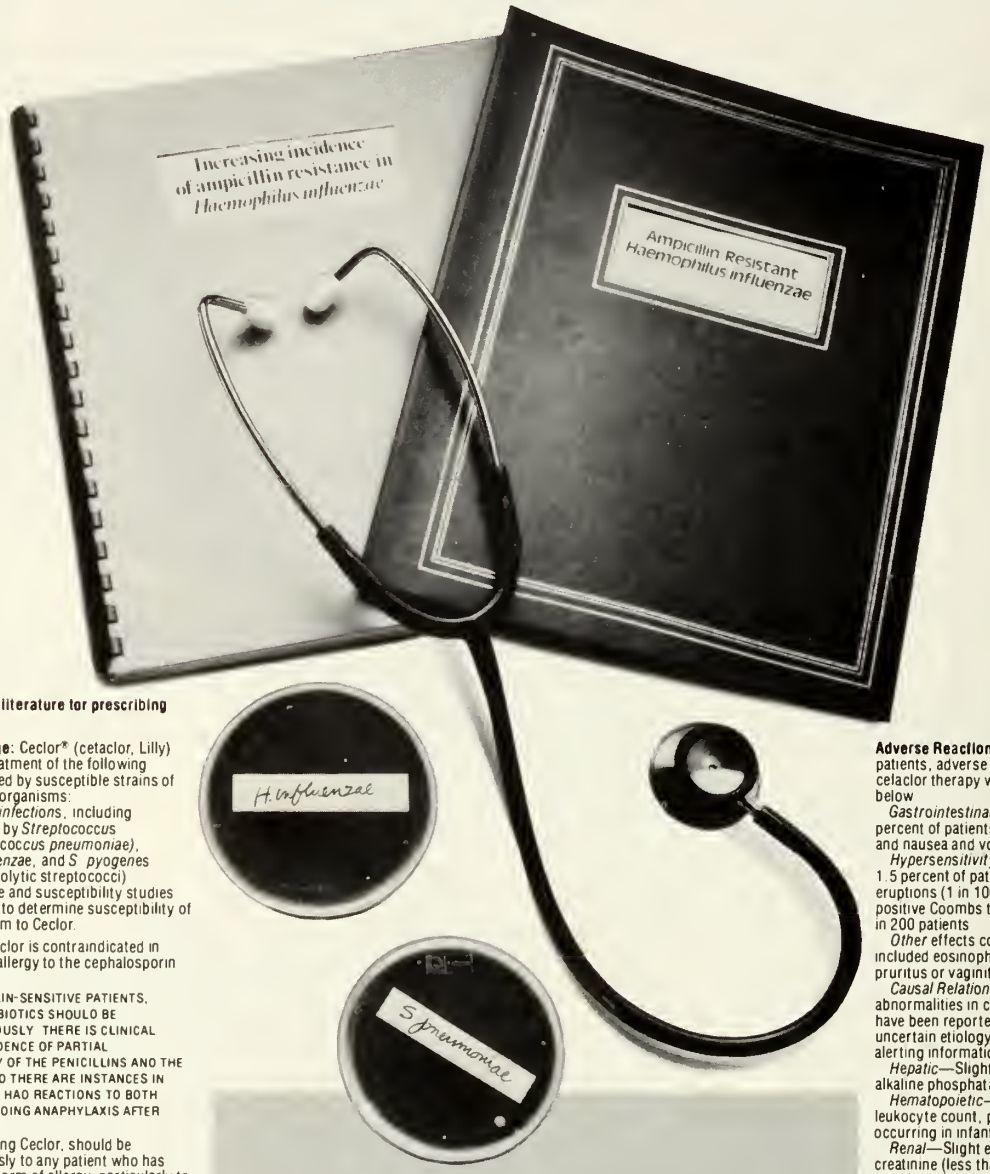
Store between 59°-86° F (15°-30° C).

Full information is available on request.

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An added complication... in the treatment of bacterial bronchitis



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

Brief Summary
Consult the package literature for prescribing information.

Indications and Usage: Ceclor* (cetaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about percent of patients and included diarrhea (1 in and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less t in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and ge pruritus or vaginitis (less than 1 in 100 patien

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests resul have been reported. Although they were of uncertain etiology, they are listed below to ser alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytos occurring in infants and young children (1 in 4

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [07

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note. Ceclor* (cetaclor) is contraindicated in patients with known allergy to the cephalosp and should be given cautiously to penicillin-a patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheu fever. See prescribing information.

References

1. Antimicrob. Agents Chemother., *8*:91, 197
2. Antimicrob. Agents Chemother., *11*:470, 1
3. Antimicrob. Agents Chemother., *13*:584, 1
4. Antimicrob. Agents Chemother., *12*:490, 1
5. Current Chemotherapy (edited by W. Siegenthaler and R. Luthy), II: 880. Washington, D.C.: American Society for Microbiology, 1978.
6. Antimicrob. Agents Chemother., *13*:861, 1
7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Disea (edited by G.L. Mandell, R.G. Douglas, Jr. J.E. Bennett), p. 487. New York, John Wil Sons, 1979.

Additional information ava the profession on request f Eli Lilly and Company, Indianapolis, Indiana 4628

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Medical School Codes

UNITED STATES

The following is a list of medical schools in the United States and Puerto Rico, existing and extinct, arranged in state order and showing the code number by which each school is designated in the geographical section of this Directory. Existing approved schools are listed in capital letters.

001 Alabama
001-02 UNIVERSITY OF ALABAMA SCHOOL OF MEDICINE, BIRMINGHAM

003 Arizona
003-01 UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE, TUCSON

004 Arkansas
004-01 UNIVERSITY OF ARKANSAS SCHOOL OF MEDICINE, LITTLE ROCK

005 California
005-02 UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE, SAN FRANCISCO
005-06 UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE, LOS ANGELES
005-11 STANFORD UNIVERSITY SCHOOL OF MEDICINE, PALO ALTO
005-12 LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE, LOMA LINDA—LOS ANGELES

007 Colorado
007-02 UNIVERSITY OF COLORADO SCHOOL OF MEDICINE, DENVER

008 Connecticut
008-01 YALE UNIVERSITY SCHOOL OF MEDICINE, NEW HAVEN

010 District of Columbia
010-01 GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-02 GEORGETOWN UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-03 HOWARD UNIVERSITY COLLEGE OF MEDICINE, WASHINGTON

011 Florida
011-02 UNIVERSITY OF MIAMI SCHOOL OF MEDICINE, MIAMI
011-03 UNIVERSITY OF FLORIDA COLLEGE OF MEDICINE, GAINESVILLE

012 Georgia
012-01 MEDICAL COLLEGE OF GEORGIA, AUGUSTA
012-05 EMORY UNIVERSITY SCHOOL OF MEDICINE, ATLANTA

016 Illinois
016-01 RUSH MEDICAL COLLEGE, CHICAGO
016-02 UNIVERSITY OF CHICAGO PRITZKER SCHOOL OF MEDICINE, CHICAGO
016-04 The Hahnemann Medical College and Hospital, Chicago
016-06 NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO
016-11 UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO
016-42 CHICAGO MEDICAL SCHOOL UNIVERSITY OF HEALTH SCIENCES, CHICAGO
016-43 LOYOLA UNIVERSITY STRITCH SCHOOL OF MEDICINE, MAYWOOD

017 Indiana
017-20 INDIANA UNIVERSITY SCHOOL OF MEDICINE, INDIANAPOLIS

018 Iowa
018-03 UNIVERSITY OF IOWA COLLEGE OF MEDICINE, IOWA CITY

019 Kansas
019-02 UNIVERSITY OF KANSAS SCHOOL OF MEDICINE, KANSAS CITY

020 Kentucky
020-02 UNIVERSITY OF LOUISVILLE SCHOOL OF MEDICINE, LOUISVILLE
020-12 UNIVERSITY OF KENTUCKY COLLEGE OF MEDICINE, LEXINGTON

021 Louisiana
021-01 TULANE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS
021-05 LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS

022 Maine
022-01 Bowdoin Medical School, Brunswick-Portland

023 Maryland
023-01 UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE, BALTIMORE
023-07 JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE, BALTIMORE

024 Massachusetts
024-01 HARVARD MEDICAL SCHOOL, BOSTON
024-07 TUFTS UNIVERSITY SCHOOL OF MEDICINE, BOSTON

025 Michigan
025-01 UNIVERSITY OF MICHIGAN MEDICAL SCHOOL, ANN ARBOR
025-07 WAYNE STATE UNIVERSITY SCHOOL OF MEDICINE, DETROIT

026 Minnesota
026-04 UNIVERSITY OF MINNESOTA MEDICAL SCHOOL, MINNEAPOLIS

027 Mississippi
027-01 UNIVERSITY OF MISSISSIPPI SCHOOL OF MEDICINE, JACKSON

028 Missouri
028-02 WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-03 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, COLUMBIA
028-20 University Medical College of Kansas City
028-22 Ensworth Medical College, St. Joseph
028-34 ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-43 Kansas City College of Medicine and Surgery
028-46 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, KANSAS CITY
028-78 Kansas City College of Osteopathy & Surgery

030 Nebraska
030-05 UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE, OMAHA
030-06 CREIGHTON UNIVERSITY SCHOOL OF MEDICINE, OMAHA
030-07 Nebraska College of Medicine, Lincoln

033 New Jersey
033-05 COLLEGE OF MEDICINE & DENTISTRY OF NEW JERSEY — NEW JERSEY MEDICAL SCHOOL, NEWARK

034 New Mexico
034-01 UNIVERSITY OF NEW MEXICO SCHOOL OF MEDICINE, ALBUQUERQUE

035 New York
035-01 COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK
035-03 ALBANY MEDICAL COLLEGE OF UNION UNIVERSITY, ALBANY
035-06 STATE UNIVERSITY OF NEW YORK AT BUFFALO, SCHOOL OF MEDICINE, BUFFALO
035-08 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, BROOKLYN
035-09 NEW YORK MEDICAL COLLEGE, NEW YORK
035-10 Bellevue Hospital Medical College, New York
035-15 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, SYRACUSE
035-19 NEW YORK UNIVERSITY SCHOOL OF MEDICINE, NEW YORK
035-20 CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK

035-45 UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE AND DENTISTRY, ROCHESTER
035-46 ALBERT EINSTEIN COLLEGE OF MEDICINE OF YESHIVA UNIVERSITY, NEW YORK

036 North Carolina
036-01 UNIVERSITY OF NORTH CAROLINA SCHOOL OF MEDICINE, CHAPEL HILL
036-05 BOWMAN GRAY SCHOOL OF MEDICINE, WINSTON-SALEM
036-07 DUKE UNIVERSITY SCHOOL OF MEDICINE, DURHAM

038 Ohio
038-02 Eclectic Medical College, Cincinnati
038-06 CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE, CLEVELAND
038-40 OHIO STATE UNIVERSITY COLLEGE OF MEDICINE, COLUMBUS
038-41 UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE, CINCINNATI
038-43 MEDICAL COLLEGE OF OHIO AT TOLEDO, TOLEDO

039 Oklahoma
039-01 UNIVERSITY OF OKLAHOMA SCHOOL OF MEDICINE, OKLAHOMA CITY

040 Oregon
040-02 UNIVERSITY OF OREGON MEDICAL SCHOOL, PORTLAND

041 Pennsylvania
041-01 UNIVERSITY OF PENNSYLVANIA SCHOOL OF MEDICINE, PHILADELPHIA
041-02 JEFFERSION MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA
041-07 MEDICAL COLLEGE OF PENNSYLVANIA, PHILADELPHIA
041-09 HAHNEMANN MEDICAL COLLEGE AND HOSPITAL, PHILADELPHIA
041-12 UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, PITTSBURGH
041-13 TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA
041-77 Philadelphia College of Osteopathic Medicine, Philadelphia

042 Puerto Rico
042-01 UNIVERSITY OF PUERTO RICO SCHOOL OF MEDICINE, SAN JUAN

045 South Carolina
045-01 MEDICAL UNIVERSITY OF SOUTH CAROLINA COLLEGE OF MEDICINE, CHARLESTON

047 Tennessee
047-05 VANDERBILT UNIVERSITY SCHOOL OF MEDICINE, NASHVILLE
047-06 UNIVERSITY OF TENNESSEE COLLEGE OF MEDICINE, MEMPHIS
047-07 MEHARRY MEDICAL COLLEGE SCHOOL OF MEDICINE, NASHVILLE

048 Texas
048-02 UNIVERSITY OF TEXAS MEDICAL BRANCH, GALVESTON
048-04 BAYLOR COLLEGE OF MEDICINE, HOUSTON
048-12 UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL, DALLAS
048-13 UNIVERSITY OF TEXAS MEDICAL SCHOOL, SAN ANTONIO

049 Utah
049-01 UNIVERSITY OF UTAH COLLEGE OF MEDICINE, SALT LAKE CITY

050 Vermont
050-02 UNIVERSITY OF VERMONT COLLEGE OF MEDICINE, BURLINGTON

051 Virginia
051-01 UNIVERSITY OF VIRGINIA SCHOOL OF MEDICINE, CHARLOTTESVILLE
051-04 MEDICAL COLLEGE OF VIRGINIA HEALTH SCIENCES DIVISION OF VIRGINIA COMMONWEALTH UNIVERSITY, RICHMOND

054 Washington
054-04 UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE, SEATTLE

055 West Virginia
055-01 WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE, MORGANTOWN

056 Wisconsin
056-05 UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON
056-06 MEDICAL COLLEGE OF WISCONSIN, MILWAUKEE

FOREIGN MEDICAL SCHOOL CODES

CANADA

060 Alberta
060-01 University of Alberta Faculty of Medicine, Edmonton

061 British Columbia
061-01 University of British Columbia Faculty of Medicine, Vancouver

062 Manitoba
062-01 University of Manitoba Faculty of Medicine, Winnipeg

065 Ontario
065-01 University of Toronto Faculty of Medicine, Toronto
065-05 Queen's University Faculty of Medicine, Kingston

067 Quebec
067-01 McGill University Faculty of Medicine, Montreal

OTHER FOREIGN

118 Afghanistan
118-01 Faculty of Medicine, Kabul University, Kabul

132 Argentina
132-01 Facultad de Ciencias Medicas de la Universidad de Buenos Aires, Buenos Aires
132-02 Facultad de Ciencias Medicas de la Universidad Nacional de Cordoba, Cordoba
132-04 Facultad de Ciencias Medicas, Farmacia y Ramos Menores de la Universidad Nacional del Litoral, Rosario, Santa Fe
132-06 Facultad de Ciencias Medicas de la Universidad Nacional de Cuyo, Mendoza, Mendoza

143 Australia
143-03 Faculty of Medicine University of Sydney, Sydney, New South Wales

154 Austria
154-07 Medizinische Fakultat der Universitat Wien, Wien (407-26 from March 13, 1938 to June, 1945)

176 Bolivia
176-03 Facultad de Medicina de la Universidad Mayor de San Simon, Cochabamba

215 Cambodia
215-01 Ecole Royal de Medicine Du Cambode, Phnompenh

231 Chile
231-01 Facultad de Medicina de la Universidad de Chile, Santiago

242 China
242 China (also see 243 Effective January 1, 1977)
242-09 St. John's University (Pennsylvania Medical School) Shanghai, Kiangsu (Extinct)
242-16 National Shanghai Medical College, Shanghai, Kiangsu
242-17 West China Union University College of Medicine and Dentistry, Chengtu, Szechuan
242-22 Aurora University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
242-39 Shansi University Medical College, Taiyuan, Shansi

243 China
243-38 National Honan University Medical College, Kaifeng, Honan (242-38 Prior to 1-1-71)
243-51 National Defense Medical Center, School of Medicine, Shanghai, Kiangsu (242-51 Prior to 1-1-71)

244 Taiwan
244 Taiwan (Formosa) effective 1-1-71
244-02 College of Medicine National Taiwan University, Taipai (385-02 Prior to 1-1-71)
244-04 Taipai Medical College, Taipai (385-04 Prior to 1-1-71)

264 Colombia
264-01 Facultad de Medicina de la Universidad Nacional de Colombia Cuidad Universitaria, Bogota, Cundinamarca
264-02 Faculdad de Medicina de la Universidad de Cartagena, Cartagena, Bolivar
264-04 Facultad de Medicina de la Pontificia Universidad Javeriana, Bogota, Cundinamarca
264-06 Facultad de Medicina de la Universidad de Caldas, Manizales, Caldas
264-07 Facultad de Medicina de la Universidad del Cauca, Popayan, Cauca

275 Cuba
275-01 Facultad de Medicina de la Universidad de la Habana, La Habana

308 Dominican Republic
308-01 Facultad de Medicina de la Universidad de Santo Domingo, Ciudad, Trujillo

319 Ecuador
319-01 Facultad de Ciencias Medicas de la Universidad Central, Quito

330 Egypt (United Arab Republic)
330-02 Kasr-el-Aini-Faculty of Medicine Cairo University, Cairo (Formerly Fouad First University Faculty of Medicine)
330-03 Faculty of Medicine Alexandria University, Alexandria

352 England
352-05 School of Medicine University of Leeds, Leeds
352-11 Registrable Qualifications granted by English Conjoint Board (Royal College of Surgeons of England/Royal College of Physicians of London)

385 Formosa (Taiwan)
385 (Also see 244 Taiwan [Effective 1-1-71])
385-01 Kaohsiung (takau) Medical College, Kaohsiung
385-02 College of Medicine National Taiwan University, Taipai
385-03 National Defense Medical Center, Taipai
385-05 China Medical College, Taichung

396 France
396-06 Faculte de Medecine de l'Universite de Paris, Paris, Seine

407 Germany
407 Also see 408-409—East and West Germany (Effective 1-1-71)
407-07 Medizinische Fakultat der Georg-August-Universitat, Gottingen, Niedersachsen
407-10 Medizinische Fakultat der Universitat Heidelberg, Heidelberg, Baden-Wurttemberg
407-15 Medizinische Fakultat der Phillipps-Universitat, Marburg/Lahn, Hessen
407-16 Medizinische Fakultat der Ludwig Maximiliams-Universitat, Munchen, Bayern
407-21 Medizinische Fakultat der Universitat Hamburg, Hamburg, Hamburg
407-23 Medizinische Fakultat der Johann-Wolfgang-Goethe-Universitat, Frankfurt-Am-Main, Hessen
407-33 Medizinische Fakultat der Freien Universitat Berlin, Berlin

409 Germany West
409-33 Medizinische Fakultat Freien Universitat, Berlin, Berlin (407-33 Prior to 1-1-71)

418 Greece
418-02 Faculty of Medicine University of Thessaloniki, Thessaloniki

451 Honduras
451-01 Facultad de Medicine y Cirugia de la Universidad Nacional Autonoma de Honduras, Tegucigalpa

473 Hungary
473-01 Orvosi Fakultas Tudomanyegyetem, Budapest

495 India (Goa)
495-01 University of Bombay, Affiliated Medical Colleges are:
a. Grant Medical College Bombay University, Bombay, Maharashtra
b. Seth Gorhandas Sunderdas Medical College Bombay University, Bombay, Maharashtra
495-04 Madras Medical College Madras University, Madras, Madras
495-08 Christian Medical College Punjab University, Ludhiana, Punjab
495-09 St. John's Medical College, Bangalore, Mysore (before June 1966: Government Medical College, Mysore University, Mysore)
495-11 Andhra Medical College Andhra University, Visakhapatnam, Andhra
495-16 Stanley Medical College Madras University, Madras, Madras
495-23 Medical College Baroda University, Baroda, Gujarat
495-27 Christian Medical College, Vellore, Madras
495-28 Byramjee Jeejeebhoy Medical College, Poona, Maharashtra
495-29 Government Medical College Punjab University, Patiala, Punjab
495-30 Sawai Man Singh Medical College Rajasthan University, Jaipur, Rajasthan
495-31 Medical College Kerala University, Trivandrum, Kerala
495-34 Gajra Rajo Medical College Vikram University, Gwalior Madhya Pradesh
495-35 Karnatak Medical College Karnatak University, Hubli, Mysore
495-36 All-India Institute of Medical Sciences, New Delhi, Delhi
495-37 Kasturba Medical College Karnatak University, Manipal, Mysore
495-41 G.S.V. Memorial Medical College Lucknow University, Kampur, Uttar Pradesh
495-47 Medical College Jabalpur University, Jabalpur, Madhya Pradesh
495-48 M.P. Shah Medical College Gujarat University, Jamnagar, Gujarat
495-49 Ghandhi Medical College Vikram University, Bhopai, Madhya Pradesh
495-54 Rajendra Medical College, Ranchi, Bihar
495-55 Sardar Patel Medical College, Bikaner, India
495-57 Kakatiya Medical College, Warangal, Andhra Pradesh
495-97 Dr. Vaishampayan Memorial Medical College, Shivaji University, Shalopur, Maharashitra

506 Indonesia
506-02 Faculty of Medicine Airlangga Airlangga University, Surabaya

517 Iran
507-01 Faculty of Medicine University of Teheran, Teheran

528 Iraq
528-01 Faculty of Medicine Baghdad University, Baghdad

539 Ireland
539-02 National University of Ireland, Constitient Colleges are:
a. Faculty of Medicine University College, Dublin
b. Faculty of Medicine University College, Cork
c. Faculty of Medicine, Galway

561 Italy
561-15 Facolta di Medicina e Chirurgia dell'Universita di Perugia, Perugia
561-19 Facolta di Medicina e Chirurgia dell'Universita di Siena, Siena

572 Japan
572-11 Tokyo Medical College (Nippon Ikadaigaku) Hongo, Tokyo (Extinct)
572-41 Faculty of Medicine Shinshu University, Matsumoto, Nagano

583 Korea (South)
583-01 Severence Medical College Yonsei University, Seoul
583-02 College of Medicine Seoul National University, Seoul
583-04 College of Medicine Kyong-Puk National University, Taegu
583-06 College of Medicine Chun Nam National University, Kwangiu
583-09 College of Medicine Pusan National University, Pusan

605 Lebanon
605-01 Medical School American University of Beirut, Beirut

649 Mexico
649-01 Facultad de Medicina de la Universidad Nacional Autonoma de Mexico, Mexico
649-02 Facultad de Medicina de la Universidad de Nuevo Leon, Monterrey, Nuevo Leon
649-06 Facultad de Medicina de la Universidad Nacional del Sureste, Merida, Yucatan
649-14 Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara Jalisco

660 Netherlands
660-61 Faculteit der Geneeskunde Universiteit Van Amsterdam, Amsterdam

671 New Zealand
671-01 Medical School University of Otago, Dunedin

704 Pakistan
704-01 King Edward Medical College, Lahore, West Pakistan
704-02 Dow Medical College, Karechi, Federal Capital Area
704-03 Dacca Medical College, Dacca, East Pakistan
704-04 Nishtar Medical College, Multan, West Pakistan
704-09 Khyber Medical College, Peshawar, North-West Frontier Province
704-10 Chittagong Medical College, Chittagong, East Pakistan (160-01 after 7-1-72)

726 Paraguay
726-01 Facultad de Medicina de la Universidad Nacional de Asuncion, Asuncion

737 Peru
737-01 Facultad de Medicina de San Fernando de la Universidad Nacional Mayor de San Marcos, Lima
737-05 Facultad de Medicina de la Universidad Nacional de San Agustin, Arequipa
737-06 Facultad de Medicina "Cayetano Heredia" de la Universidad Peruana de Ciencias Medicas y Biologicas, Lima

748 Phillipines
748-01 Faculty of Medicine and Surgery University of Santo Tomas, Manila
748-02 College of Medicine University of the Phillipines, Manila
748-07 College of Medicine Manila Central University, Manila
748-08 Institute of Medicine Far Eastern University, Manila
748-09 College of Medicine Southwestern University, Cebu City
748-10 College of Medicine University of the East, Quezon City

803 Scotland
803-01 Faculty of Medicine University of Aberdeen, Aberdeen
803-03 Faculty of Medicine University of Edinburgh, Edinburgh

836 South Africa
836-01 Medical School University of the Witwatersrand, Johannesburg

847 Spain
847-04 Facultad de Medicina de la Universidad de Madrid, Madrid
847-08 Facultad de Medicina de la Universidad de Valencia, Valencia
847-10 Facultad de Medicina de la Universidad de Salamanca, Salamanca

869 Switzerland
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869-02 Medizinische Fakultat der Universitat Bern, Bern
869-05 Faculte de Medecine de l'Universite de Lausanne, Lausanne

875 Syria
875-01 Faculty of Medicine Damascus University, Damascus

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891 Thailand
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891-02 Faculty of Medicine at Sariraj Hospital University of Medical Sciences, Thonburi

902 Turkey
902-01 Tip Fakultesi Istanbul Universitesi, Istanbul

913 Union of Soviet Socialist Republics
913-02 Voronez Medical Institute, Voronez

957 Yugoslavia
957-02 Medicinski Fakultet Univerziteta u Beogradu, Beograd

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RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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George M. Penn, M.D., Topeka (913) 234-9566
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
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Kermit Wedel, M.D., Minneapolis (913) 392-2144

Kansas Medical Society, Topeka (913) 235-2383/235-3619

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CARRO, F AURELIO, WINFIELD
CARSON, RICHARD CARLYLE, PITTSBURG
CARTER, MACK A, WICHITA
CARVER, LARRY A, TOPEKA
CASADY, GILBERT N, HUTCHINSON
CASEY, JAMES, HUTCHINSON
CASEY, JOHN J, WICHITA
CASHMAN JR, MAURICE R, TOPEKA
CASTEEL, CHARLES K, KANSAS CITY
CATHCART-RAKE, WILLIAM F, SALINA
CATHEY, ROBERT H, MANHATTAN
CAUBLE, WILBUR G, WICHITA
CAUGHRON, MICHAEL ROBERT, SHAWNEE MISSION
CAVANAUGH, CLAIR J, GREAT BEND
CAVANAUGH, JOHN W, TOPEKA
CAVITT, ROBERT F, SHAWNEE MISSION
CAWLEY, LEO P, WICHITA
CECIL III, JOHN, HAYS
CEDERLIND, CRANSTON JAY, SHAWNEE MISSION
CENAC, MARK T, LEAVENWORTH
CHAFFEE, DEAN C, ABILENE
CHALIAN, ALEXANDER R, KANSAS CITY
CHAMBERLIN JR, CECIL R, TOPEKA
CHANEY, ERNIE J, BELLEVILLE
CHANG, CHAE H, KANSAS CITY
CHANG, FREDERIC C, WICHITA
CHANG, SHU FANG, SHAWNEE MISSION
CHAPMAN, JAMES H, WICHITA
CHAPPUIE, WILLIAM G, INDEPENDENCE
CHARO, FREDERICK H, WICHITA
CHAVALA, SUDARSAN, ARKANSAS CITY
CHEDIAK, ELIAS, LAWRENCE
CHEN, TAK-MING, TOPEKA
CHENOWETH, JOHN R, O.D., OLATHE
CHERRY JR, ARTHUR C, TOPEKA
CHERVEN, PHILIP L, HUTCHINSON
CHEUNG, P W H, TOPEKA
CHIN, TOM D, KANSAS CITY
CHO, CHENG T, KANSAS CITY
CHO, SECHIN, WICHITA
CHONKO, ARNOLD M, KANSAS CITY
CHOTIMONGKOL, ANUPONG, DODGE CITY
CHOW, STANLEY Y, FORT SCOTT
CHOY, JAMES K L, TOPEKA
CHRISTENSEN, MARION O, KIOWA
CHRISTIAN, STANLEY J, SHAWNEE MISSION
CHRISTMAN JR, CARL, WICHITA
CHRONISTER, BERT, NEODESHA
CHUBB, RICHARD M, BAXTER SPRINGS
CHUN, CHUNG S, KANSAS CITY
CHUNG, JOHN J, SHARON SPRINGS
*CIFUENTES, RAUL F, WICHITA
CISKEY, WILLIAM J, EUREKA
CLAASSEN, MILTON A, NEWTON
CLARK, COURTNEY, WICHITA
CLARK, CRAIG N, TOPEKA
CLARK, DAVID H, SALINA
CLARK, LAURENCE A, WAMEGO
CLARK, ORVILLE R, ST PETERSBURG,FL
CLARK, RAY A, LAKE CHAS,LA
CLARK, ROBERT THOMAS, GUATEMALA CEN A,
CLEAVER, EDGAR M, WICHITA
CLENDENIN, ROBERT KEELE, SHAWNEE MISSION
CLIFTON, H DAVID, WICHITA
CLINTON, DALE L, LAWRENCE
CLYDE, HARRIE R, TEMPE,AZ
COALE, LLOYD H, KANSAS CITY
COBB, LESLIE H, MULVANE
COCHRAN, PAUL W, TOPEKA
CODY, DOROTHY, HAYS
CODY, JOHN, HAYS
COE, RICHARD D, SHAWNEE MISSION
COFFEY, ROY B, SALINA
*COHEN, JUSTIN THOMAS, WICHITA
COHEN, LOUIS, TOPEKA
COHEN, ROBERT A, SHAWNEE MISSION
COHLMIA, JERRY B, WICHITA
COHN, STEVEN G, SHAWNEE MISSION
COHNBERG, ROSELLEN E, CEDAR VALE
COKELEY, JOHN M, TOPEKA
COLOSMITH, DONALD C, EMPORIA
COLE, WARD M, WELLINGTON
COLEMAN, GARY, ABILENE
COLEMAN, THOMAS J, WICHITA
COLIP, F MERLYNN, NORTON
COLLIER, HAROLD W, WICHITA
COLLIER, WILLIAM J, MCPHERSON
COLLINS, DEAN T, TOPEKA
COLLINS, EDWARD JOSEPH, TOPEKA
COLLINS, FRANCIS T, TOPEKA
COLLINS, FRANK BUSH, WINFIELD
COMBS, G RALPH, LEAVENWORTH
COMBS, PETER S, LEAVENWORTH
CONARD, CLAIR C, DODGE CITY
CONCEPCION JR, EUGENIO S, WICHITA
CONNELLY, JOHN C, TOPEKA
CONNELLY, MAURICE R, SALINA
CONNOR, BRIAN, SALINA
CONRARDY, PETER A, WICHITA
CONROY, ROBERT W, TOPEKA
COOK, DONALD RAY, WICHITA
COOK, G EDWARD, WICHITA
COOK, JAMES O, KANSAS CITY
COOKE, ALLAN R, KANSAS CITY
COOMER, TYLER E, PITTSBURG
COOPER, ARTHUR E, NORTON
COOPER, JACK R, SHAWNEE MISSION
COOPER, KENT J, PITTSBURG
COOPER, LEO F, SHAWNEE MISSION
COPELAND, GARY A, JUNCTION CITY
COPELAND, SHARON LAVARNE, KANSAS CITY
COPENING, TELL B, IOLA
CORBIN, MURRAY O, SHAWNEE MISSION
CORDELL, LARRY D, KANSAS CITY, MO
CORDER, ROBERT L, HIGHLAND
CORDER, S SCOTT, OTTAWA
CORRIGAN, GEORGE F, WICHITA
COSSMAN, F PRICE, WICHITA
COTTON, ROBERT T, TOPEKA
COULTER, HENRY F, SHAWNEE MISSION
COULTER, THOMAS B, KANSAS CITY,MO
COVERT, THOMAS J, SALINA
COWLES, GORDON T, WICHITA
COX, JACK A, EMPORIA
COX, ROBERT H, HAYS
COX III, IRA L, KANSAS CITY
COX JR, IRA, SHAWNEE MISSION
COX JR, WALLACE F, KANSAS CITY
COYLE, JOHN F, COFFEYVILLE
CRAIG, CHARLES C, NEWTON
CRAM, ERNEST R, ST FRANCIS
CRAM JR, OLE R, LARNED
CRAMER, GUY W, PARSONS
CRAMM, RUSSELL E, HAYS
CRANE, C HERBERT, MANHATTAN
CRANE, DAVID D, WICHITA
CRANSTON, STEPHEN D, NEWTON
CRARY, JOHN E, TOPEKA
CRAWFORD, ROBERT A, HUTCHINSON
CRIST, ROBERT D, KANSAS CITY, MO
*CRISWELL, MILORED LOUISE, WICHITA
CROCKETT, CHARLES A, KANSAS CITY
CRONEMEYER, RICHARD L, KANSAS CITY,MO
CRONIN, DONALD J, WICHITA
CROUCH, CAPT STEVEN W, DOVER,DE
CROUCH, WILLIAM H, TOPEKA
CROW, ERNEST W, WICHITA
CROWLEY, EDWARD X, WICHITA
CULP, LOUIS M, KANSAS CITY
CULTRON, FRANK T, SALINA
CULVER, WARREN T, LAWRENCE
CUMMINGS, RICHARD J, WICHITA
CURRAN, KEVIN E, SHAWNEE MISSION
CURTIS, MIRJANA R, KANSAS CITY

D

D'SOUZA, BISMARCK C, SALINA
DAEHNKE, SIGURD S, WINFIELD
DAHL, ASHER W, COLBY
DALUM, PETER JOSEPH, CLAY CENTER
DANIELS, ROBERT M, WICHITA
DARR, RICHARD B, KANSAS CITY
DAUGHERTY, ROBERT M, MEADE
DAVENPORT, S SCOTT, TUSCALOOSA,AL
DAVIDSON, HARRY T, WICHITA
DAVIS, CHESTER R, TOPEKA
DAVIS, CHRISTOPHER G, KANSAS CITY
DAVIS, DAVID H, LARNED
DAVIS, DAVID R, EMPORIA
DAVIS, GEORGE R, ELLSWORTH
*DAVIS, PAUL H, WICHITA
DAVIS, RICHARD E, SHAWNEE MISSION
DAVIS, RONALD B, WICHITA
DAVIS JR, JAMES W, KANSAS CITY,MO
*DAY, HOWARD, WICHITA
DAY, HUGHES W, KANSAS CITY
DE BAKKER, JAN B, WICHITA
DE BRIERE, SIDNEY L, PARSONS
DE SMET, ARTHUR AUGUST, KANSAS CITY
DE SOIGNE, RAPHAEL R, TOPEKA
DE SOUZA, DERRICK J, LEAVENWORTH
DECHAIRO, THOMAS, WESTMORELAND
DECKER, DONALD D, HALSTEAD
DECKERT, ROSALIE E, WICHITA
DEGNER, JAMES B, GREAT BEND
DEITZ, MICHAEL R, KANSAS CITY
DEJONG, DAVID C, WICHITA
DELGADO, SERGIO, TOPEKA
DELLETT, KENNETH B, EL DORADO
DELP, MAHLON H, SHAWNEE MISSION
DELPHIA, ROBERT E, OLATHE
DEMOSS, ELEANOR P, WICHITA
DEMOTT, WAYNE R, KANSAS CITY
DENISON, TERRY R, SHAWNEE MISSION
DEPENBUSCH, FRANCIS L, HUTCHINSON
DEPOE, JOSEPH H, WINFIELD
DERRINGTON, KENNETH L, SHAWNEE MISSION
DETAR, GEORGE F, IOLA
DIACON, JAMES L, WELLINGTON
DIALLO, GASTON I, KANSAS CITY
DIAMENT, MORTON LEONARD, MARION
DICK, ARTHUR R, KANSAS CITY
DICK, WILLIS G, IOLA
DICK JR, HENRY J, CHANUTE
DICKINSON, CHARLES R, COFFEYVILLE
DIEDERICH, DENNIS A, KANSAS CITY
DIEHL, ANTONI M, KANSAS CITY,MO
DIENER, CLAYTON H, HESSTON
DILL, ROONEY SIDNEY, ATWOOD
DIVER, ROBERT W, BILOXI,MS
DIXON, RAYMOND W, COFFEYVILLE
DLABAL, FRANK A, WILSON
DOBRATZ, ROBERT A, BELOIT
DOCKHORN, ROBERT J, SHAWNEE MISSION
DOHERTY, WILLIAM R, SHAWNEE MISSION
*DOLAN JR, PHILIP JARVIS, WICHITA
DONAHOE, DORVAL H, MEADE
*DONATELLE, EDWARD P, WICHITA
DONNELL, JAMES M, WICHITA
DONNELLY, FRANCIS M, KANSAS CITY
DOORNBOS, J FRED, WICHITA
DOUBEK, HERBERT D, BELLEVILLE
DOUGHERTY, THOMAS M, GARNETT
DOUGLAS, JOSEPH MAHLON, OLATHE
DOWELL, JAMES C, SALINA
DOWNING, GREGORY, WICHITA
DOZIER, FRED S, HERINGTON
DRAEMEL, H RICHARD, SALINA
DRAKE, DOUGLAS J, BELOIT
DRAKE, RALPH L, WICHITA
DREHER, HENRY S, SALINA
DRENICK, FRANCES EKEC SE, PITTSBURG
DREVETS, CURTIS C, WICHITA
DRIGGS, GUY K, WICHITA
DUCKETT, THOMAS G, HIAWATHA
DUCKETT II, THOMAS G, SHAWNEE MISSION
DUICK, GREGORY, WICHITA
DUJOVNE, CARLOS A, KANSAS CITY
DUNAGIN, JACK A, TOPEKA
DUNLAP, RICHARD L, LAWRENCE
DUNN, DANIEL R, SCOTT CITY
DUNN, MARVIN I, KANSAS CITY
DUNSHEE, CARLYLE M, FORT SCOTT
DURAND, ANTONIO C, WICHITA
DURKEE, WILLIAM R, MANHATTAN
DURST JR, ROBERT D, TOPEKA
DUYSAK, SAMI, LEAVENWORTH
DVORAK, EDUARD, KANSAS CITY
DWORZACK, DAVID L, WICHITA
DYCK, ARTHUR H, MCPHERSON
DYCK, CORA E, WICHITA
DYCK, GEORGE, NEWTON
DYER, VERNON E, WICHITA

E

EASTES, GARY DEAN, HALSTEAD
EATON, GLEN E, SALINA

* Probationary members.

EATON, LESLIE F, SALINA
ECKART, DE MERLE E, HUTCHINSON
ECKERT, WILLIAM G, WICHITA
EDDY, VICTOR M, HAYS
EDROZO, M LUZ LUNA, COFFEYVILLE
EDWARDS, DAVID J, EMPORIA
EDWARDS, MANIS C, WICHITA
EFTEKHARI, SAIED, WICHITA
EGEA, FERNANDO M, KANSAS CITY
EGELHOF, RICHARD H, WICHITA
EICHHORN, FRANK O, GARDEN CITY
EIDT, DAVID W, OLATHE
EIDT, LAURENCE A, OLATHE
EIKERMANN, WILLIAM C, SHAWNEE MISSION
ELDER, DOUGLAS M, TOPEKA
ELLEDGE, E FRED, GREAT BEND
ELLIS, HARVEY D, WICHITA
ELLIS, STEPHEN S, COFFEYVILLE
ELLISON, PAUL D, SALINA
ELNEN, WALTER T, WICHITA
EMERY, FRANK A, WICHITA
EMMOT, WILLIAM W, INDEPENDENCE
EMPSON, CHARLES L, INDEPENDENCE
ENBERG, ROBERT, HAYS
ENDERS, WRAY, SHAWNEE MISSION
ENNS, EUGENE K, NEWTON
ENNS, JAMES H, NEWTON
ENRIQUEZ, ROMAN, HOWARD
ENS, GERHARD GEORGE, HILLSBORO
ENS, PETER, HILLSBORO
ENSEY, T CRANFORD, MARION
EPLEE, JOHN P, WICHITA
ERICKSON, CLARENCE W, PITTSBURG
ERKEN, RONALD V, WICHITA
ESCH, JOHN G, PITTSBURG
ESRIG, HAROLD L, O.D., SHAWNEE MISSION
ESTES, NORMAN CLEON, KANSAS CITY,MO
ETZENHOUSER III, RUSSELL O, SHAWNEE MISSION
EVANS, FARRIS O, WICHITA
EVANS, GRANT E, WICHITA
EVANS, KENNETH L, SHATTUCK,OK
*EVANS, ROGER WILLIAMS, WICHITA
EVANS, WILLIAM R, GREAT BEND
EVANS JR, WILLIAM E, SHAWNEE MISSION
EWING, THOMAS O, LARNED
EYSTER, ROBERT L, WICHITA

F

FABIAN, CAROL J, KANSAS CITY
FAIRCHILD, JOHN A, MANHATTAN
FALTER, RICHARD T, HUTCHINSON
FARHA, GEORGE J, WICHITA
FARHA, S JIM, WICHITA
FARRAR JR, WILLIAM H, ARKANSAS CITY
FAST, ROBERT E, ATCHISON
FAST, W SPENCER, ATCHISON
FEAGAN, JERRY, TOPEKA
FEIGHNY, ROBERT E, SAN ANTONIO,TX
FELT, SAMUEL E, SHAWNEE MISSION
FENDER JR, THOMAS H, WICHITA
FENT, LEE S, NEWTON
FENTON, ROBERT M, GARDEN CITY
FERGUSON, ROBERT LEON, SHAWNEE MISSION
FERNANDEZ, LUIS A, TOPEKA
FERNIE, ROBERT W, HUTCHINSON
FERREE, RICHARD ALLAN, MCPHERSON
FERRIS, BRUCE G, WICHITA
FESTOFF, BARRY W, KANSAS CITY,MO
FEUILLE JR, EDMOND G, WICHITA
FIELD, RICHARD A, TOPEKA
FIELDS, GALEN W, SCOTT CITY
*FIELDS, STEPHEN A, WICHITA
FIESER, CARL W, GREAT BEND
FILLEY, VERNON W, PRATT
FILLMAN, ELDON M, TOPEKA
FINK, ABRAHAM A, PLANTATION,FL
FINLEY, DENNIS R, WICHITA
FIRKINS, RICHARD T, DES MOINES,IA
FISCHER, REX R, MANHATTAN
FISHER, JAMES B, WICHITA
FITZGERALD, EDWARD J, WICHITA
FITZGERALD, JAMES E, GOODLAND
*FITZIG, SANFORD, WICHITA
FITZPATRICK, M ROBERT, KANSAS CITY
FIXLEY, MARK S, KANSAS CITY
FLANDERS, H ALDEN, HAYS
FLECKENSTEIN, CHARLES S, ONAGA
FLEMING, FORNEY W, WICHITA
FLOERSCH, HUBERT M, KANSAS CITY
FLOWERS JR, CLELL B, WICHITA
FORD, CHARLES R, WICHITA
FORD, FRED L, TOPEKA
FORDYCE, NORMAN, SHAWNEE MISSION
FORET, JOHN O, KANSAS CITY
FORRED JR, WALTER A, HARPER
FORSTER JR, LOUIS G, SALINA
FORTUNE, CEDRIC B, OLATHE
FOSS, DANIEL C, HUTCHINSON
FOSTER, CHARLES G, TOPEKA
FOSTER, D BERNARD, TOPEKA
FOWLER, ROBERT J, WICHITA
FOWLER, WAYNE L, CONCORDIA
FOX, DEANNA K, KANSAS CITY
FOX, HOWARD A, KANSAS CITY
FRANCIS, NORTON L, WICHITA
FRANCISCO, CLARENCE L, SHAWNEE MISSION
FRANCISCO, EDGARDO, HORTON
FRANCISCO, W DAVID, KANSAS CITY
FRANSEN, HERBERT, NEWTON
FRANSEN, PAUL H, NEWTON
FRANZ, ROBERT G, HILLSBORO
FRAZIER, RICHARD L, EMPORIA
FREDERICK, M F, HUGOTON
FREDRICKSON, DUANE E, LINDSBORG
FREEBORN JR, WARREN S, CLYDE
FREEMAN, F GILES, PRATT
FREEMAN, FRED A, MANHATTAN
FREEMAN, RAYMOND S, SALINA
FRIESEN, DALE, LAWRENCE
FRIESEN, FLORENCE V, HESSTON
FRIESEN, ORLANDO J, BUHLER
FRIESEN, STANLEY R, KANSAS CITY
FRIGGERI, ROBERT W, GIRARD
FRITZ, GEORGE E, WICHITA
FRITZEMEIER, WILLIAM H, WICHITA
FROMM, ARTHUR H, WICHITA
FRY, LUTHER L, GARDEN CITY
FULLEN, JERYL G, SHAWNEE MISSION
FULLER, DERYL O, LINDSBORG
FULTON, JOHN K, WICHITA
FUNK, EDWARD D, LINWOOD
FUSILLO, MICHAEL, TOPEKA

G

GAINES, LARRY STRAWDER, CHARLESTON,SC
GALICHIA, JOSEPH P, WICHITA
GALLEHUGH, KEITH W, SHAWNEE MISSION
GALVAN, ALONSO, WICHITA
GANDHI, SHANTIKUMAR K, TOPEKA
GANN, E LAMONTE, EMPORIA
GANS, FREDERICK A, SALINA
GANZARAIN, RAMON C, TOPEKA
GARCIA, FRANCISCO, SHAWNEE MISSION
GARCIA, GOULO C, EMPORIA
GARDINER, TED M, GARDEN CITY
GARDNER, BILLIE L, HARPER
GARDNER, GLENN M, SHAWNEE MISSION
GARDNER, JAMES D, MANHATTAN
GAREY, WILLIAM JOHN, INDEPENDENCE
GATENO, JOSEPH, GREAT BEND
GAY, JOHN O, TOPEKA
GEHRT, EARL B, CHANUTE
GEITZ, JAMES M, EMPORIA
GELVIN, E RAYMOND, CONCORDIA
GENCH, RAYMOND L, CARMEL,CA
GENDEL, JOSEPH E, TOPEKA
GENGELBACH, RICHARD W, WICHITA
GENILO, AMANCIO C, WICHITA
*GENILO, CELESTE A, WICHITA
GENTRY, KALE C, SHAWNEE MISSION
GEORGE, EARL F, WICHITA
GEORGE, M DON, WICHITA
*GERBER, ALLEN D, WICHITA
GERBER, HARRY A, LEAVENWORTH
GERJARUSAK, PRAPAS, KANSAS CITY
GESSLER, DONALD J, WICHITA
GETTLER, DEAN T, FORT SCOTT
GIAP, HAI PHUC, KANSAS CITY
GIBBS, EUGENE, COFFEYVILLE
GIESSEL, MICHAEL D, TOPEKA
GILBERT, J HOWARD, SENECA
GILBERT, ROBERTA M, SHAWNEE MISSION
GILBERT II, JOHN H, GARDEN CITY
GILHOUSEN, FREDERIC M, KANSAS CITY
GILL, GEORGE L, LAMPE,MO
GILLEN, BILLY A, SHAWNEE MISSION
GILLES, HELEN M, LAWRENCE
*GILMARTIN, RICHARD C, WICHITA
GILMORE, CLARENCE A, KANSAS CITY
GIMPLE, KENNETH, TOPEKA
GINAVAN, DUANE A, EMPORIA
GIROD, CHARLES I, EL DORADO
GIVNER, DAVID, WICHITA
GLAZZARD, CHARLES O, OLATHE
GLEASON, JIMMIE A, TOPEKA
GLENN, JAMES N, EMPORIA
GLENN, LYLE G, PROTECTION
GLOVER, RICHARD M, NEWTON
GNAU, FREDRIC B, HALSTEAD
GODFREY, KENNETH E, TOPEKA
GODFREY, ROBERT G, KANSAS CITY,MO
GODFREY, WILLIAM A, KANSAS CITY
GODWIN, PHILLIP A, LAWRENCE
GOERING, DONALD D, COLDWATER
GOERING, EMIL L, TOPEKA
GOERING, ROBERT C, WICHITA
GOERTZ, KENNETH K, KANSAS CITY
GOERTZ, LEO R, KANSAS CITY
GOHIL, MAHENDRA N, WICHITA
GOLDBERG, HERBERT R, WICHITA
GOLLERKERI, MOHAN P, SHAWNEE MISSION
GOLLIER, ROBERT A, OTTAWA
GOLLIER II, ROBERT A, OTTAWA
GOMEZ, FRANCISCO, SHAWNEE MISSION
GOMEZ, MODESTO S, PITTSBURG
GONZALEZ, FRANCISCO, WICHITA
GONZALEZ, HIRAM, WICHITA
GOOD, JAMES T, FORT SCOTT
GOOD, WENDELL LISLE, SHAWNEE MISSION
GOODIN, ETHELYNN A, ALEXANDRIA,LA
GOODIN, WALKER D, ALEXANDRIA,LA
GOODPASTURE, HEWITT C, WICHITA
GOODPASTURE, WILLARD C, HUTCHINSON
GOODWIN, DONALD W, KANSAS CITY
GOOTEE, JOSEPH E, TOPEKA
GOTO, HIROSHI, KANSAS CITY
GOULDNER, RENE M, WICHITA
GOYLE, KRISHAN K, WICHITA
GOYLE, VIMAL, WICHITA
GRADINGER, BILLENS C, *,AR
GRADY, KENNETH L, KANSAS CITY
GRAHAM, J MALCOLM, LEAVENWORTH
GRAHAM, KENNETH L, LEAVENWORTH
GRAHAM, THOMAS W, LEAVENWORTH
GRAHAM JR, CHARLES P, TOPEKA
GRANTHAM, JARED J, KANSAS CITY
GRAUEL, CHARLES W, WICHITA
GRAVES, JACK W, WICHITA
GRAVES, KATHRYN, HUTCHINSON
GRAY, C LUCIEN, WICHITA
GRAY, CAPTAIN KING, LAWRENCE
GRAY, DAVID E, TOPEKA
GRAY, H TOM, WICHITA
GRAYIB, ANTOINE S, TOPEKA
GRAYSON, ROY D, ALBUQUERQUE,NM
GREEN, LAWRENCE C, ARKANSAS CITY
GREENBERG, GEORGE E, DODGE CITY
GREENBERG, MARK, TOPEKA
GREENBERGER, N J, KANSAS CITY
GREENE, HORACE T, TOPEKA
GREENWOOD, EDWARD O, TOPEKA
GREENWOOD, JAMES F, ULYSSES
GREER, JAMES A, WICHITA
GREER, RICHARD H, TOPEKA
GRIBBLE, ROBERT N, WICHITA
GRIFFING, RICHARD B, SALINA
GRIFFITH, FRANK H, SALINA
GRIGSBY, KENNETH R, SUN CITY,AZ
GRIGSBY JR, GEORGE T, TOPEKA
GRILLOT, FLOYD B, WICHITA
GRIMALDI, GARY A, FORT SCOTT
GRIMES, I ROSS, LIBERAL
GRIMES, JAMES T, LYONS
GRIPKEY, CLARENCE A, KANSAS CITY
GRISOLIA, ANDRES, LEAVENWORTH
GRISWOLD, DALE G, NEWTON
GROHS, HEINZ K, WICHITA
GROVE, JOHN A, NEWTON
GROWNEY, JOHN T, ATCHISON
GRUENDEL, RICHARD A, KANSAS CITY
GRUENDEL, VIRGINIA T, KANSAS CITY
GRUND, FRANK M, WICHITA
GRUSHNYS, ARNOLD, WICHITA
GSELL, GEORGE F, WICHITA
GUERRA, TOMAS H, MARION
GUMUCIO, MARIO L, BOLIVIA,
GUNN, MARVIN R, SALINA
GUNTER, CARL C, QUINTER
*GUNTHER, LORETTA ANN, WICHITA
GUTHRIE, RICHARD A, WICHITA
GUTOVITZ, ALLEN LOUIS, TOPEKA
GUZMAN, MANUEL, SALINA

H

HA, SANG W, COFFEYVILLE
HAAS, CHARLES O, KANSAS CITY
HAAS, JUDITH S, SHAWNEE MISSION
HACKER, DAVID CHARLES, SHAWNEE MISSION
*HACKER, ELAINE MARY, TOPEKA
HADLEY, DELMONT C, OTTAWA
HAFFNER, WILLIAM N, EL DORADO
HAGAN, C THOMAS, WICHITA
HAGAN, FRANCIS J, WICHITA
HAGGAN, MARGARET E, LAWRENCE
HAIGLER, JAMES P, HAYS
HAINES, CHESTER W, HAVEN
HAINES, ROBERT A, TOPEKA
HALE, RALPH, WICHITA
*HALL, J ROGER, WICHITA
HALL, JERRY D, ANTHONY
HALL, WESLEY H, GIRARD
HALL OO, KENDALL WM, GARDNER
HALL III, THOMAS BRYAN, KANSAS CITY
HALLEY, M MARTIN, TOPEKA
HALLING, L WILLIAM, HAYS
HALPIN, EDWARD D, WICHITA
HALVORSON, HOWARD C, OLATHE
HAMILTON, JAMES J, WAKEENEY
HAMILTON, NORMAN G, TOPEKA
HAMM, ORVAL L, PAKISTAN,
HAMMEKE, JOHN C, LEAVENWORTH
HAMMEL, GEORGE W, BELLA VISTA,AR
HAMTIL, LAWRENCE W, SHAWNEE MISSION
HAN, CHAN S, COFFEYVILLE
HANCOCK, ALAN C, KANSAS CITY
HANDLEY, DENNIS MICHAEL, SHAWNEE MISSION
HANSA, HANSA, HAYS
HANSON, DAVID C, IOLA
HARA, GLENN S, KANSAS CITY
HARBIN, GARY LYNN, SALINA
HARD, BENJAMIN F, KANSAS CITY, MO
HARDIN, CREIGHTON A, KANSAS CITY
HARMS, ALBERT C, SHAWNEE MISSION
HARMS, EDWIN M, WICHITA
HARMS, WILMER A, HALSTEAD
HARPSTER, GENE O, SHAWNEE MISSION
HARRIS, FRANK H, WICHITA
HARRIS, HUBERT L, TOPEKA
HARRIS, NORMAN R, SALINA
HARRIS, NORVAN D, LIBERAL
HARRIS, PATRICIA A, TOPEKA
HARRIS JR, CLAIB B, GARNETT
HARRISON, A BROOKS, WICHITA
HARRISON, HALL E, TOPEKA
*HARRISON, PAUL BARRY, WICHITA
HART, DILLIS L, WICHITA

* Probationary members

HART, LAWRENCE E, ATCHISON
HARTLEY, FOUNT K, HONOLULU,HI
HARTLEY, ROY WILEY, NORTON
HARTMAN, CHARLES R, KANSAS CITY
HARTMAN, GERALD V, KANSAS CITY
HARTMAN, ROGER L, NORTON
HARTOCOLLIS, PETER, TOPEKA
HARTONG, WILLIAM ALLEN, SHAWNEE MISSION
HARVEY, JOHN E, EMPORIA
HARVEY, ROSEMARY B, WICHITA
HARWOOD, CLAUDE J, GLASCO
HASKINS, ROBERT J, CHANUTE
HASSELLE III, JAMES E, LAWRENCE
HASSETT, GERARD R, COLBY
HASSLER, RANDY D, SALINA
HASTINGS, MARY T, SHAWNEE MISSION
HATHAWAY, PETER, SHAWNEE MISSION
HATTON, DONALD W, LAWRENCE
HATTON, LLOYD W, SALINA
HATTRUP, RICHARD J, WICHITA
HAVENHILL II, MARSHALL A, EMPORIA
HAWLEY, RAYMOND G, WICHITA
HAYES, JAMES PATRICK, LYNCH,NE
HAYES, KRIS A, LYONS
HAYES, WILLIAM L, WICHITA
HAYS, THOMAS H, WICHITA
HEASTY, ROBERT G, MANHATTAN
HEBBAR, SATYA N, TOPEKA
HEDRICK, KENNETH E, HUTCHINSON
HEINS, LAWRENCE G, ABILENE
HENDERSON, CHARLES F, PARSONS
HENDERSON, JOHN M, ALBUQUERQUE,NM
HENNEBERRY, MICHAEL, HUTCHINSON
HENNING, CALVIN W, OTTAWA
HENNING, CHARLES E, WICHITA
HENNING JR, HAROLD JOHN, KANSAS CITY
HENRY, JOSEPH E, SHAWNEE MISSION
HENSLEY JR, CLINE D, WICHITA
HERED, JOHN, WICHITA
HERMES, RICHARD L, LAWRENCE
HERMRECK, ARLO S, KANSAS CITY
HERRERA, JORGE J, TOPEKA
HERSHORN, SIMON E, WICHITA
HERZON, CHARLES D, SHAWNEE MISSION
HESSE, FREDERICK J, SALINA
HESSE, ROBERT S, TULSA,OK
HESSER, HERBERT H, SHAWNEE MISSION
HIEBERT, ABRAHAM E, WICHITA
HIEBERT, DAVID L, LAWRENCE
HIEBERT, JOHN B, TOPEKA
HIEBERT, PETER E, SHAWNEE MISSION
HIESTERMAN, HERMAN W, QUINTER
HILOYARD, VICTOR H, COLBY
HILOYARD II, VICTOR H, COLBY
HILL, JAMES E, BELLA VISTA,AR
HILL, JOHN J, COLORADO SPRING,CO
HILL, PHILLIP K, HALSTEAD
HILL, RICHARD H, MEADE
HILL, ROBERT N, TOPEKA
*HILST, WILBUR DEAN, TOPEKA
HINSHAW, ALFRED H, WICHITA
HINSHAW, CHARLES T, WICHITA
HINSHAW, EDGAR D, ARKANSAS CITY
HINTHORN, DANIEL R, KANSAS CITY
HIRATZKA, TOMIHARU, WICHITA
HIRO, WAYNE E, LAWRENCE
HIRSCHBERG, J COTTER, TOPEKA
HISZCZYNSKYJ, ROMAN, TOPEKA
HITCHCOCK, C THOMAS, KANSAS CITY
HIZON, RAMON R, WICHITA
HOBBS, DONALD D, TOPEKA
HOBSON, MILBURN W, SHAWNEE MISSION
HODES, HERBERT C, SHAWNEE MISSION
HODGES, BRUCE E, SHAWNEE MISSION
HODGES, GLENN R, KANSAS CITY
HODGES, MERLE A, SALINA
HODGSON, DAVID K, WASHINGTON
*HODGSON, ROBERT B, WICHITA
HODSON, HERVEY R, WICHITA
HOFER, OEWAYNE O, CONCORDIA
HOFFER, JOHN G, MEDICINE LODGE
HOHERZ, DAVID G, TOPEKA
HOLCOMB, DONALD G, LOS ANGELES,CA
HOLCOMB, WILLIAM M, LIBERAL
HOLOCRAFT, JACQUELYNE, KANSAS CITY
*HOLDEN, DAVID M, WICHITA
HOLDEN JR, RAYMOND F, WICHITA
HOLDERMAN, WALLACE D, HUTCHINSON
HOLLADAY, HARMON M, WICHITA
HOLLADAY, KENNETH R, EUDORA
HOLLEMAN JR OD, JAMES F, KANSAS CITY
HOLMAN, JON B, SALINA
HOLMES, FREDERICK F, KANSAS CITY
HOLMES, GRACE E, KANSAS CITY
HOLMES, JED, WICHITA
HOLSINGER, DONALD M, PITTSBURG
HOLT, BEVLEY DAN, MANHATTAN
HOLT, JOHN M, GREAT BEND
HOLT, ROBERT G, HALSTEAD
HOOFER, WILFORD D, HALSTEAD
HOOGSTRATEN, BARTH, KANSAS CITY
HOPKINS, WILLIAM O, SHAWNEE MISSION
HOPKINS JR, B MORRISON, SCOTT CITY
HOPKINS JR, LENLY T, SHAWNEE MISSION
HOPPER, CHARLES R, EMPORIA
HORBELT, DOUGLAS V, WICHITA
HORNE, JAMES B, TOPEKA
HORSEMAN, ROBERT F, SHAWNEE MISSION
HORTON, WILLIAM A, KANSAS CITY
*HOSTETTER, JAMES P, TOPEKA
HOSTETTER, PHILIP H, MANHATTAN
HOUSHOLDER, DANIEL FAIR, WICHITA
HOUSHOLDER, MARTHA S, WICHITA
HOUSTON II, LAWRENCE MORLEY, SHAWNEE MISSION
HOWARD, DONALD O, WICHITA
HOWERTER JR, BERNARD E, COFFEYVILLE
HOYLE, WINTHROP E, ESTES PARK,CO
HOYT, ARTHUR W, TOPEKA
HSU, CHENG H, TOPEKA
HSU, SHIN-FU, TOPEKA
HUAMAN, ANTONIO M, TOPEKA
HUDSON, JAMES R, CHANUTE
HUDSON, ROBERT P, OLATHE
HUEBERT, DEAN A, WICHITA
HUEBNER, ROBERT STEPHAN, PITTSBURG
HUERTER, DAVID F, PITTSBURG
HUERTER, QUENTIN C, KANSAS CITY
HUGHES, ROBERT WALTER, LAWRENCE
HULL, KENNETH L, WICHITA
HULTGREN, MYRON K, WICHITA
HUME, JOSEPH W, WICHITA
HUMMER, LLOYD M, WICHITA
HUMPHREY, LOREN JENKINS, SHAWNEE MISSION
HUNNINGHAKE, RONALD E, MINNEAPOLIS
HUNTER, KENNETH R, LEBO
HUNTER JR, JAMES S, MANHATTAN
HURWITZ, ARYEH, KANSAS CITY
HUSEMAN, RICHARD ALLAN, NORTH KANS CITY,MO
HUSTEAD, ROBERT F, WICHITA
HUSTON, FRANCIS W, WINCHESTER
HUSTON, JOSEPH W, TOPEKA
HUTCHINSON, OIRK T, WINFIELD
HUTCHISON, GLEN C, HAYS
HUTTON, FREDERICK A, TOPEKA
HWA, EUGENE C, NEWTON
HYDE, MARSHALL E, FT SMITH,AR
HYLWA, THEODORE M, SHAWNEE MISSION
HYNES, HARRY E, WICHITA

I

IBARRA, J LUIS, WICHITA
IBARRA, RICHARD C, KANSAS CITY
*IDBEIS, BADR, WICHITA
*ILORETA, ALFREDO TABYTOL, TOPEKA
INGHAM JR, H LAIRD, LAWRENCE
INGRAM, JOHN E, KANSAS CITY
INNES, ROBERT C, KANSAS CITY
*IOANNOU, NICHOLOAS, WICHITA
IRBY, ADDISON C, FORT SCOTT
IRBY, PRATT, FORT SCOTT
ISAAC, CHARLES A, NEWTON
ITURRALDE, GEORGE, SHAWNEE MISSION
IWAY, BELIND D, ELKHART

J

JABEL, JUVENAL T, SATANTA
JACKMAN, GLENN H, ESTES PARK,CO
JACKS, J WARREN, PRATT
JACKSON, CHARLES R, WICHITA
JACKSON, LINDA H, TOPEKA
JACKSON, ROGER PAUL, N KANSAS CITY,MO
JACKSON, VICTOR L, ALTAMONT
JACKSON JR, DELMAS A, SALINA
JACOB, KANNAMPALLY L, EL DORADO
JACOBS, DAVID S, KANSAS CITY
JACOBS, RAE R, KANSAS CITY
JACOBSEN, DWIGHT SKINNER, COLBY
JACOBY II, ROBERT E, TOPEKA
JAHANIAN, DARYOUSH, KANSAS CITY
JAMBOR, JAMES J, DODGE CITY
JAMES, VERNON L, WICHITA
JANES, DONALD R, SHAWNEE MISSION
JANSSEN, ERWIN T, TOPEKA
JANZEN, HERMAN F, HILLSBORO
JARROTT, JOHN B, HUTCHINSON
JAYARAM, MARANDAPALLI R, KANSAS CITY
JAZAYERLI, NABIL, WICHITA
JEFFRIES, RHONDA DETERT, KANSAS CITY
JEHAN, SAYED S, WICHITA
JENKINS, EDWARD, GARDEN CITY
JENKINS, NEAL M, SALINA
JENNEY, CHARLES B, WICHITA
JENSEN, THOMAS M, OLATHE
JESTER, SHELBY L, WICHITA
JEWELL, ROSS L, ST FRANCIS
JEWELL, WILLIAM R, KANSAS CITY
JOHLER, TERRY HARTWIG, LEAVENWORTH
JOHNS JR, LEO E, KANSAS CITY,MO
*JOHNSON, EDEN E, TOPEKA
JOHNSON, FREDERICK E, TOPEKA
JOHNSON, GARY L, WICHITA
*JOHNSON, GEORGE K, WICHITA
JOHNSON, HOWELL O, DODGE CITY
JOHNSON, J RICHARD, MCPHERSON
JOHNSON, JOHN E, KANSAS CITY
JOHNSON, NADINE KAY, SHAWNEE MISSION
JOHNSON, PAUL D, LEAVENWORTH
JOHNSON, THOMAS E, WICHITA
JONES, CHARLES E, SHAWNEE MISSION
*JONES, CLIFFORD E, WICHITA
JONES, EDWARD L, GREAT BEND
JONES, FORREST H, COLUMBUS
JONES, H IVOR, SHAWNEE MISSION
JONES, H PENFIELD, LAWRENCE
JONES JR, HERMAN H, KANSAS CITY
JOSEPH, BRIAN W, TOPEKA
JOSEPH, HOWARD F, LAWRENCE
JOSS, CHARLES S, TOPEKA
JOUVENAT, NEIL C, SHAWNEE MISSION
JOYCE, G BERNARD, TOPEKA
JUBELT, HILBERT P, MANHATTAN
JUDILLA JR, FRANCISCO, WICHITA
*JULIUS, SATISH CHANDRA, TOPEKA
JUSTUS, WILLIAM J, PLEASANTON

K

KADIAN, RAJESH S, SHAWNEE MISSION
KADISON, HERBERT I, WICHITA
KAGAN, STUART M, SHAWNEE MISSION
KALBAC, RICHARD W, GARDEN CITY
KALOOR, RICHARD M, MANHATTAN
KALIVAS, JAMES T, KANSAS CITY
KANE JR, WILLIAM M, HAYS
KAROATZKE, E STANLEY, WICHITA
KAROATZKE, JON K, WICHITA
KASHA, ROBERT L, WICHITA
KASHYAP, BANSHI PRASAD, SHAWNEE MISSION
KASSEBAUM, GLEN E, EL DORADO
KASSEBAUM, KENNETH G, WICHITA
KASSEL, HENRY W, LAKEWOOD,CO
KATZ, JEROME B, TOPEKA
KAUFMAN, EUGENE E, WICHITA
KAUFMAN, LELAND R, WINFIELD
KAUFMAN, WILLARD E, MOUNDRIDGE
KAUL, ANAND N, WINFIELD
KAVEL, KARL K, TOPEKA
KAYLOR, MARIETTA J, STAFFORD
*KEARNS, NORBERT W, TOPEKA
KEENE, GEORGE H, WICHITA
KEENY, M GARY, WICHITA
KEITH, ROBERT MARSHALL, EMPORIA
KELLER, JAMES P, WICHITA
KELLING, COLLYER, EMPORIA
KELLY, DAN A, TOPEKA
KELLY, ROBERT W, WICHITA
KEMPTHORNE, CHARLES R, MANHATTAN
KENDALL, TOM E, WICHITA
KENDRICK, J GILLERAN, WICHITA
KENNEDY, GERALD T, WICHITA
KENNEDY, HOWARD U, TOPEKA
KENNEDY, JAMES A, KANSAS CITY
KENNEDY, KENNETH R, SHAWNEE MISSION
KEPES, JOHN J, KANSAS CITY
KERBY, GERALD R, KANSAS CITY
KESTENBAUM, THELDA M, KANSAS CITY
KETCHUM, LYNN O, SHAWNEE MISSION
KEYS JR, ROBERT C, TOPEKA
KEYS SR, ROBERT C, TOPEKA
KHICHA, GYANCHAND J, WICHITA
KHOURY, GEORGE H, WICHITA
KHURANA, SATISH K, SHAWNEE MISSION
KIFER, C JAMES, HAYS
KIHM, ALBERT A, CHANUTE
KIM, JONG M, KANSAS CITY
KIM, PAIK N, WICHITA
KIM, YONG W, TOPEKA
KIMBALL, RICHARD R, MANKATO
KIMBLE, JAMES A, WICHITA
KIMURA, CHARLES C, SHAWNEE MISSION
KINOLING, PAUL H, TOPEKA
KING, CHARLES R, KANSAS CITY
KING, WILLIAM T, GREAT BEND
KINNAN, L F, CALDWELL
KIRBY, MERLIN G, GREAT BEND
KIRCHMER, NANCY, KANSAS CITY
KIRCHNER, FERNANDO R, KANSAS CITY
KIRK, THOMAS E, MANHATTAN
KIRK JR, E DAVID, WICHITA
KIRKEGAARD, ROGGER S, TOPEKA
KISER, JOHN L, WICHITA
KISER, WILLARD J, WICHITA
KITCHEN, ROBERT R, WICHITA
KLASSEN, DANIEL S, NEWTON
KLEINHOLZ JR, EMIL JOHN, TOPEKA
KLEMMER, HERBERT, TOPEKA
KLENDA JR, MARTIN B, BELOIT
KLIEWER, VERNON L, NEWTON
KLINGLER JR, EUGENE A, MANHATTAN
*KLONTZ, WILLIAM JOSEPH, WICHITA
KLOSTERHOFF, BRUCE E, HUTCHINSON
KNAPP, LESLIE E, WICHITA
KNAPP, M ROBERT, WICHITA
KNAPPENBERGER, ROY C, WICHITA
KNECHT, STEPHEN M, EMPORIA
KNEIDEL, THOMAS W, WICHITA
KNUTH, KENNETH L, INDEPENDENCE
KODANAZ, A AYTEKIN, SHAWNEE MISSION
KOKSAL, TOM, GARDEN CITY
KOONS, JESS W, LIBERAL
KOSAR, CLARENCE D, CONCORDIA
KOURI, SAMMY H, WICHITA
KOVARIK, ERNEST O, TOPEKA
KOZIKOWSKI, BEN M, SHAWNEE MISSION
KRANTZ, KERMIT E, KANSAS CITY
KRAUSE, ROLAND L, WICHITA
KREHBIEL, MARK A, SALINA
KROLL, HARRY G, TOPEKA
KROLL, RICHARD, FT HUACHEICA,AZ
KRUCKEMYER, ALAN L, SALINA
KRUEGER, HAVEN C, GREAT BEND
KRUEGER, KURT ALLEN, SHAWNEE MISSION
*KRUPKA, JOHN J, WICHITA
*KRUPKA, MILES ALBERT, HAYSVILLE
KUBIN, DORIS A, SHAWNEE MISSION
*KUBINA, GLENN RICHARD, WICHITA
KUMAR, SURINDER, NEWTON
KUNNAWUTHIDEE, KAMOLTIPYA, DODGE CITY

* Probationary members.

KURTH, C JOSEPH, WICHITA
KURTH, ROBERT H, SHAWNEE MISSION
KUTILEK, FRANK J, WICHITA
KWEE, SIOE T, KANSAS CITY
KYNER, JOSEPH L, KANSAS CITY

L

LABHSETWAR, S A, JUNCTION CITY
LACEY, STEFFAN R, WICHITA
LAFENE, BENJAMIN W, MANHATTAN
LAHAM, ALEXANDER J, AUGUSTA
LAI, JENG Y, WICHITA
LAING, ROBERT R, KANSAS CITY
LAIRO, OALE O, OLATHE
LAKE, MAX S, SALINA
*LAMBERT, EUGENE KENT, WICHITA
LANCE, RAYMOND W, PITTSBURG
LANCE JR, JOHN F, WICHITA
LANSKY, LESTER L, KANSAS CITY
LANSKY, SHIRLEY B, KANSAS CITY
LAPI, RUTH M, SHAWNEE MISSION
LARSON, OELBERT L, HIAWATHA
LASLEY, OAVID A, SALINA
LASLEY, MICHAEL B, HAYS
LATIMER, KATHERINE, WICHITA
LAURY, OAVID G, OTTAWA
LAUVER, MARY ANN, WICHITA
LAVA, CHIRUND, PARSONS
LAW, FINOLEY, ELLINWOOD
LAWLESS, HAROLD L, BLUE RAPIDS
LAWN, CLAUDIA A, WICHITA
LAWN, RAYMOND A, WICHITA
LAWRY, JAMES VORIS, WINFIELD
LAWS, LEWIS R, MARYSVILLE
LAWSON, DWIGHT, TOPEKA
LAWSON, ROBERT C, TOPEKA
LAWTON, MARVIN K, CONCORDIA
LAYBOURNE JR, PAUL C, KANSAS CITY
LAZAR, HARRY, WICHITA
LEAKE III, HUNTER C, KANSAS CITY
LEARNED, GEORGE R, LAWRENCE
LEATHERS III, HOLLIS K, SHAWNEE MISSION
LEE, JAE M, KANSAS CITY
LEE, KYO R, KANSAS CITY
LEE, R REX, WICHITA
LEE, SANG OUG, FT DODGE, IA
LEE, SONG DOW, TOPEKA
LEE, SONG PING, TOPEKA
LEE, YONG U, EL DORADO
LEE JR, EDWARD S, WICHITA
LEE JR, JAMES G, KANSAS CITY
LEFFLER, PAUL B, PITTSBURG
LEGASPI JR, PEDRO L, SHAWNEE MISSION
LEGER, LEE H, FT MYERS, FL
*LEGLER, GARY LEE, TOPEKA
LEIGH, LAWRENCE E, SHAWNEE MISSION
LEIKER, JOSEPH, ARKANSAS CITY
LEISY, JERALD W, WICHITA
LEITCH, DAVID A, GARNETT
LEMOINE JR, ALBERT N, KANSAS CITY
LENEVE, ROBERT T, HUGOTON
LENSKI JR, FRANCIS X, IOLA
LENTZ, WILLIAM R, TOPEKA
LEO, WILLIAM A, KANSAS CITY
LESSENDEN, GLENN A, LAWRENCE
LESSENDEN JR, C M, TOPEKA
LESTER, JOHN BUCKLES, SHAWNEE MISSION
LETTNER, HANS T, HUTCHINSON
LEVINE, ERROL, KANSAS CITY
LEVINE, WILLIAM R, WICHITA
LEVY, EDWIN Z, TOPEKA
LEWIN, WALTER, SHAWNEE MISSION
LEWIS, JAMES E, SHAWNEE MISSION
LEWIS JR, H DANIEL, KANSAS CITY, MO
LIEBERMAN, BRUCE IRWIN, KANSAS CITY
LIES, BARTHEL N, COLWICH
LIES, JAMES E, WICHITA
LIES, RICHARD B, WICHITA
LIM, CARLO, SEDAN
LIN, JOE J, WICHITA
LIN, MAU SHONG, TOPEKA
LINDLEY, MILTON E, WICHITA
LINDSLEY, CAROL B, KANSAS CITY
LINDSLEY, HERBERT B, KANSAS CITY
LINHARDT, RONALD O, WICHITA
LINSHAW, MICHAEL A, KANSAS CITY
LIPSEY, JAMES H, SHAWNEE MISSION
LISKOW, BARRY IRWIN, KANSAS CITY
LITTLE, L GILBERT, WICHITA
LIU, CHIEN, KANSAS CITY
LIUJULUSCHAN, RIT, PARSONS
LIVINGSTON, CHARLES E, SALINA
LLOYD, HARVEY L, KANSAS CITY
LLOYD, JAMES W, CONCORDIA
LOCKHART, JOSEPH G, WICHITA
LOEFFLER, JAMES A, WICHITA
LOEWEN, HENRY H, WICHITA
LOEWEN, PETER S, HILLSBORO
LOEWEN, WILLIAM C, WICHITA
LOFGREEN, VICTOR J, OTTAWA
LOGAN, GEOFFREY G, WICHITA
LOGANBILL, VARDEN J, MOUNDRIDGE
LOHMEYER, KENNETH L, EMPORIA
LONEY, JOHN M, BELOIT
LONG, EDWARD E, HUMBOLDT
LONG, JOHN W, TOPEKA
LONG, LLOYD O, GOODLAND
LONG, ROBERT C, NORTON
LONGWOOD, ORLIN W, TUCSON, AZ
LOPEZ, RUBEN ENRIQUE, GOESSEL
LORCH, VICHIEN, WICHITA
LOREN, MICHAEL LEWIS, SHAWNEE MISSION
LOTITO, CARLOS A, KANSAS CITY
LOVELAND, G CHARLES, LAWRENCE
LOVETT, PAUL A, WICHITA
LOW, HAROLD L, WICHITA
LOW, JOSEPH H, SUN CITY, AZ
LOWE, DEWITT S, DUARTE, CA
LOWE, STANLEY W, MANHATTAN
LOWMAN, JAMES T, KANSAS CITY
LUEDTKE, WALTER E, EMPORIA
LUEGER OD, JAMES JOHN, SENECA
LUEKEN, LUEKE B, WICHITA
LUELLEN, THOMAS J, WICHITA
LUETJE, CHARLES MARION, KANSAS CITY, MO
LUKENS, DAVID, HUTCHINSON
LUKERT, BARBARA P, KANSAS CITY
LULO, ANTONIO R, SHAWNEE MISSION
LUMB, RAYMOND C, TOPEKA
LUNGSTRUM, JACK E, SALINA
LUZZATI, ENZO F, WICHITA
LYNCH, JOHN A, TOPEKA
LYNCH, SEAN R, KANSAS CITY
LYONS JR, FRANK C, MANHATTAN

M

MAC KILLOP JR, DANIEL, WINFIELD
MACARTHUR, RICHARD IAN, KANSAS CITY
MACE, RONALD O, JUNCTION CITY
MACY, NORMAN E, SALINA
MACY, TED L, SALINA
MADDEN, WILLIAM J, LINCOLN
MADISON, WILLARD K, NORTONVILLE
MADISON JR, WARD N, WICHITA
MADSEN, GLENN L, LAWRENCE
MAGIDSON, ELLIOT ARTHUR, WICHITA
MAGILL, ALFRED MORTON, TOPEKA
MAGSALIN, ROMULO, WICHITA
MAKLAD, M NABIL, KANSAS CITY
*MALIK, MUMTAZ ILAHI, WICHITA
MALLORY, JOHN A, SHAWNEE MISSION
MALONE, EUGENE M, HALSTEAD
MANAHAN, G EUGENE, LAWRENCE
MANGOLD, JOEL JOYCE, KANSAS CITY
MANI, MANI M, KANSAS CITY
MANLEY, JOSEPH W, SHAWNEE MISSION
MANNING, ROBERT T, WICHITA
MANSFIELD, CARL M, KANSAS CITY
MANSOUR, BADIE S, WICHITA
MANTZ, FRANK A, SHAWNEE MISSION
MARCHBANKS, DONALD L, SALINA
MARSH, CONNIE M, HALSTEAD
MARSH, HENRY O, WICHITA
MARSHALL, B M, TOPEKA
MARSHALL, GEORGE O, COLBY
MARSHALL, GEORGE W, SALINA
MARSHALL, RONALD L, MANHATTAN
MARTIN, DANIEL C, MANHATTAN
MARTIN, EARL A, PARSONS
MARTIN, NORMAN L, KANSAS CITY
MARTIN, OLIVER L, SALINA
MARTIN, WILLIAM O, TOPEKA
MARTIN JR, ALBERT E, BELLA VISTA, AR
MARTIN JR, GLEN E, WICHITA
MARVIN, NORMAN G, KANSAS CITY
MARYMONT JR, JESSE H, WICHITA
MASER, GEORGE R, SHAWNEE MISSION
MASTERS, FRANCIS W, KANSAS CITY
MASTERSON, BYRON J, KANSAS CITY
MASTERSON, MELVIN LEROY, TROY
MASTIO JR, GEORGE J, WICHITA
MATASSARIN, BENJAMIN M, WICHITA
MATASSARIN, FREDERICK W, WICHITA
MATHEWS, ROBERT MAJOR, SHAWNEE MISSION
MATHEWSON, HUGH S, KANSAS CITY
MATHIS, JERRY L, SALINA
MATTHEW, WILLIAM L, OLATHE
MATTICK, IRVIN H, HAYS
MATTIOLI, LEONE, KANSAS CITY
MAU, WALTER, TOPEKA
MAUCK, HAROLD C, STOCKTON
MAWDSLEY, MICHAEL W, WICHITA
MAXFIELD, RUSSELL J, GARDEN CITY
MAXWELL, GORDON E, SALINA
MAXWELL, ROBERT A, SHAWNEE MISSION
MAY, KENNETH L, BONNER SPRINGS
MC NICKLE, GEORGE ANDREW, ASHLAND
MCALLASTER, WENDALE E, GREAT BEND
MCBRIDE, ANN, WICHITA
MCCANN, PATRICK E, FORT SCOTT
MCCANN, WILLIAM E, OLATHE
MCCARTER, DUANE K, TOPEKA
MCCARTHY, ROBERT P, KANSAS CITY
MCCAUGHEY, HUGH W, SHAWNEE MISSION
MCCLANAHAN, WARD A, WICHITA
MCCLELLAN, ERNEST L, WICHITA
MCCLELLAN, JOHN W, TOPEKA
MCCLURE, JAMES A, TOPEKA
MCCOLLUM, MARY B, LEAVENWORTH
MCCOLLUM, WILLIAM B, LEAVENWORTH
MCCOMAS JR, MARMADUKE O, CONCORDIA
MCCORMICK, EUGENE C, WELLINGTON
MCCOY, CHARLES P, WICHITA
MCCOY, CHARLES T, HUTCHINSON
MCCOY, RONALD, DODGE CITY
MCCRAE, SPENCER C, SALINA
MCCULLOUGH, ROBERT C, GOODLAND
MCEACHEN, WILLIAM H, SHAWNEE MISSION
MCELHINNEY, CHARLES F, DODGE CITY
MCELROY, MICHAEL R, SHAWNEE MISSION
MCELROY, ROBERT T, TOPEKA
MCELROY, WILBUR J, TOPEKA
MCGINNIS, MICHAEL J, DODGE CITY
MCGOVERN, JAMES L, WELLINGTON
MCGUIRE, WILLIAM F, WICHITA
MCGURK, THOMAS E, SHAWNEE MISSION
MCKEE, DICK B, PITTSBURG
MCKEE, LEO F, COTTONWOOD FALLS
MCKEE, RICHARD S, LEAVENWORTH
MCKENNA, MICHAEL J, FORT SCOTT
MCKENZIE, STEVE LEWIS, CHERRYVALE
MCKERRACHER, ROBERT O, DERBY
MCKIM, W LYNN, KINSLEY
MCKNIGHT, DAVID E, MANHATTAN
MCKNIGHT, ELLIS B, ALMA
MCLAIN, KENNETH, RANSOM
MCMAHON, MERRI M, WICHITA
MCMULLEN, JOSEPH E, HUTCHINSON
MCNEIL, ELBERT D, MANHATTAN
*MCREYNOLDS, CHARLES R, WICHITA
MCVAY, R BRUCE, CLAY CENTER
MCWHERTER, LOTTIE B, SHAWNEE MISSION
MEADOWS, DONALD C, WICHITA
MEBUST, WINSTON K, KANSAS CITY
MEDEARIS SR, DONALD N, KANSAS CITY
MEDINA, ANIBAL, HAYS
MEE, ADRIAN W, OLATHE
MEEK, GEORGE C, ARKANSAS CITY
MEEK, PALMER F, MANHATTAN
MEEK JR, JOSEPH C, KANSAS CITY
MEEKER II, BRUCE P, WICHITA
MEGIBOW, ALAN O, TOPEKA
MEHAFFY, ORVILLE A, BAXTER SPRINGS
MEHRA, PROMILLA, WICHITA
MEHRHOF, EDWARD G, CHENEY
MEIDINGER, RAY, HIAWATHA
MEIDINGER, RICHARD, TOPEKA
*MELEAN, JAIME, WICHITA
MELENDRES, JUANITO M, OLATHE
MELHORN, J MARK, KANSAS CITY
MELIA JR, B JAMES, SHAWNEE MISSION
MENAKER, JEROME S, WICHITA
MENDIONES, L MARLENE, WICHITA
MENDIONES, RUPERTO D, WICHITA
MENEHAN, H JAMES, WICHITA
MENEZ, CESAR V, SHAWNEE MISSION
MENKING, F W MANFRED, WICHITA
*MENKING, SUSAN MARGARET, WICHITA
MENNINGER, KARL A, TOPEKA
MENNINGER, ROBERT G, TOPEKA
MENNINGER, ROY W, TOPEKA
MENNINGER, W WALTER, TOPEKA
MERCADER, MARIO S, WICHITA
MEREDITH, W TOM, WICHITA
MERKEL, EARL D, RUSSELL
MERRITT, JOE P, WICHITA
MERRITT, W HENRY, LEAVENWORTH
MERSHON, JAMES C, WICHITA
MESINA, ROLANDO R, KANSAS CITY
MEULBROEK, HARVEY J, WICHITA
MEYER, WARREN E, WICHITA
MEYERS, STEPHEN, GARDEN CITY
MICHELBACH, ALBERT P, WICHITA
MIGUELINO, OLIVER M, EMPORIA
MIH, ALEXANDER, CHANUTE
*MILFELD, DOUGLAS J, WICHITA
MILITANO, THOMAS CHARLES, KANSAS CITY
MILLER, ABRAHAM H, MANHATTAN
MILLER, CHARLES H, PARSONS
MILLER, CLYDE W, WICHITA
*MILLER, DAVID PATERSON, WICHITA
MILLER, DEAN M, PARSONS
MILLER, DON E, WICHITA
MILLER, EARL E, PITTSBURG
MILLER, ELDEN V, SALINA
MILLER, FRANKLIN R, WINFIELD
MILLER, FREEMAN LANCE, SHAWNEE MISSION
MILLER, HERBERT C, NORFORD, CT
MILLER, LAWRENCE H, DERBY
MILLER, MONTE B, ANDREWS AFB, MD
MILLER, PHILIP A, WICHITA
MILLER, ROBERT E, GARDEN CITY
MILLER, STEPHEN FRANCIS, PARSONS
MILLIGAN, DONALD B, OLATHE
MILLS, CHARLES O, WICHITA
MILLS, GEORGE QUINTON, WICHITA
MILLS JR, PHILIP E, TOPEKA
MIMOSO, JOSE J, DODGE CITY
MINER JR, PHILIP B, KANSAS CITY
MINNICK, CHARLES V, JUNCTION CITY
MIRZA, MEDO, WICHITA
MITCHELL, ALEX C, LAWRENCE
MITCHELL, JOHN C, SALINA
MITTLEMAN, FREDERICK S, TOPEKA
MITTS, ERNEST W, BONNER SPRINGS
MODDRELL, CAROL A, LAWRENCE
MODLIN, HERBERT C, TOPEKA
MOELLER, DONALD D, KANSAS CITY
MOFFAT, ROBERT E, KANSAS CITY
MOHLER, JACK M, ABILENE
MONCKTON, LAWRENCE A, LAWRENCE
MONTGOMERY, LLOYD DAN, HALSTEAD
MONTGOMERY, THOMAS ALLEN, SABETHA
MONTGOMERYSHORT, RUTH G, HALSTEAD
MOORE, DENNIS F, WICHITA
MOORE, HUGH C, TOPEKA

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MOORE, JAMES E, CONCORDIA
MOORE, ROBERT, HOISINGTON
MOORE, ROBERT F, CANEY
MOORE, WAYNE V, KANSAS CITY
MOORHEAD JR, F ALLEN, NEODESHA
MORALES, AMALIA O, OSAWATOMIE
MORALES, OTTO E, OSAWATOMIE
MORANTZ, ROBERT A, SHAWNEE MISSION
MORFFI, RAUL R, KANSAS CITY
MORGAN, DICK A, WICHITA
MORGAN, JAMES I, WICHITA
MORGAN, JOHN L, EMPORIA
MORGAN, NOVA L, HAYSVILLE
MORGAN II, DAVID LLOYD, OLATHE
MORGAN III, LOUIS S, WICHITA
MORONEY, JEAN M, SHAWNEE MISSION
MORRIS, MERLE D, TOPEKA
MORRISON, IRA R, ATCHISON
*MORRISON, MICHAEL R, TOPEKA
MORRISON, RICHARD A, KANSAS CITY
MORRISON, RICHARD L, ELLINWOOD
MORROW, THOMAS F, WICHITA
MORROW JR, J TARLTON, TOPEKA
MORTON, JOHN E, HALSTEAD
MOSELEY, JACK E, WICHITA
MOSER, ERNEST C, HOLTON
MOSER, M ROSS, HOLTON
MOSER, ROY H, HOLTON
MOSIER, STANLEY JAY, WICHITA
MOSIER, STEVEN J, MANHATTAN
MOU, BIN, BAXTER SPRINGS
MOWERY, WILLIAM E, SALINA
MOWRY, GERALD L, MANHATTAN
MOYER, HERMAN J, DERBY
MOYER, JOHN D, TOPEKA
MUEHLBERGER, JAMES J, SHAWNEE MISSION
MUELLER, ARNOLD V, TOPEKA
MUELLER, J KENT, SHAWNEE MISSION
MUELLER, VERNETTE A, WICHITA
MULL, JOHN C, HUTCHINSON
MULLEN JR, CLIFFORD J, KANSAS CITY
MULLEN SR, CLIFFORD J, KANSAS CITY
MULLER, SAMUEL B, PITTSBURG
MULLINIX, JANICE M, WICHITA
MURFITT, MALCOLM C, LINDSBORG
MURPHY, BARRY L, WICHITA
MURPHY, DUANE A, WICHITA
MURPHY, PAUL M, WICHITA
MURPHY, THOMAS MEAD, TOPEKA
MURRAY, KENT B, WICHITA
*MYERS, AGNES E, WICHITA
MYERS, JO ANN, TOPEKA
MYERS, ROBERT W, NEWTON
MYERS, W EUGENE, IOLA
MYERS JR, EARL B, INDEPENDENCE
MYRICK, MICKEY, HAYS

N

NABOURS, RICHARD D, TOPEKA
NACHTIGALL, ANDREW, NEWTON
NAGESH, KAVI G, LEAVENWORTH
NAIK, GOPAL V, PADLA
NALDOZA JR, FAUSTINO M, WELLINGTON
NANCE, JOEL, TOPEKA
NARCISO, VICENTE D, ABILENE
NASH, NEWMAN CURTIS, SCOTTSDALE, AZ
NASH, ROBERT A, SHAWNEE MISSION
NAUER, PAULA LOU, SHAWNEE MISSION
NEEL, WILBUR B, HUTCHINSON
NEFF, JAMES R, KANSAS CITY
NEIBURGER, JAMES B, SHAWNEE MISSION
NEIGHBOR, ERNEST G, KANSAS CITY
NEIGHBOR, ERNEST H, KANSAS CITY
NEIGHBOR, GAYLORD P, KANSAS CITY
NEIGHBOR, RALPH M, EMPORIA
NEIL, ROY NEWTON, HAYS
NEIS, HARRY B, OSAGE BEACH, MO
NELSON, BRYAN C, SHAWNEE MISSION
NELSON, GERALD D, WICHITA
NELSON, JAMES N, TOPEKA
NELSON, PAUL L, CONCORDIA
NELSON, RICHARD D, LAWRENCE
NELSON, RUSSELL ALAN, WICHITA
NELSON, T EUGENE, FORT SCOTT
NELSON, WILLIAM PAUL, KANSAS CITY
NELSON JR, GUST H, WICHITA
NEMMERS, DAVID J, WINFIELD
NESMITH, LESLIE W, WICHITA
NEUENSCHWANDER, JOHN, HOXIE
NEUENSCHWANDER, JOHN RAND, HOXIE
NEUER, FREDERICK S, EMPORIA
NEUSCHAFER, DARREL R, HUTCHINSON
NEVINS, RICHARD L, LIBERAL
NEVITT, J RUSSELL, MORAN
NEWBY, JAMES P, WICHITA
NEWCOMB, WARD M, HAYS
NEWKIRK, DARREL D, KANSAS CITY
NEWMAN, CLIFFORD B, PITTSBURG
NEWSOM, F CARTER, WICHITA
NEWTON, CHARLES R, VISALIA, CA
NIBBELINK, LARRY WAYNE, KANSAS CITY
NICE, G WILLIAM, TOPEKA
NICHOLS, ROBERT R, FORT SCOTT
NICKELL, WAITSTILL B, SALINA
NICKELL, WENDELL K, SALINA
NIEDEREE, W CURTIS, GREAT BEND
NIEMAN, JOHN L, SHAWNEE MISSION
NIENSTEDT, JOHN F, SUN CITY, AZ
NIKON, RICHARD R, CONCORDIA
NIKON, WILLIAM A, WICHITA
NOHE, PHILIP C, KANSAS CITY
NORRIS, CHARLEY W, KANSAS CITY
NORRIS, ROBERT P, WICHITA
NORTH, DORIS G, WICHITA
NORTH, VICTOR, WICHITA
NORTON, ROBERT K, WICHITA
NOSTI, JUAN C, SHAWNEE MISSION
NOTHNAGEL, ARNOLD F, KANSAS CITY
NOVOTNY, PETER C, TOPEKA
NOWLIN, NANCY S, WICHITA
NULL, WILLIAM G, SALINA
NUNEMAKER, MARION E, HUTCHINSON
NUNEZ, JULIAN, KANSAS CITY
NYBERG, FREDRIK F, WICHITA
NYE, C ERIK, SHAWNEE MISSION
NYSTROM, CURTIS A, TOPEKA

O

O'BOYNICK II, PAUL LEONARD, KANSAS CITY
O'BRYAN, JAMES J, SHAWNEE MISSION
O'CONNELL, FRANK A, SHAWNEE MISSION
O'DONNELL, HAROLD F, ELLSWORTH
O'DONNELL, HARRY E, JUNCTION CITY
O'DONNELL JR, LEONARD A, WICHITA
O'DONNELL SR, LEONARD A, WICHITA
O'DONNELL, RICHARD H, CLAY CENTER
O'GRADY, JOSEPH A, GARFIELD, AR
O'NEIL, ROBERT H, TOPEKA
O'SHEA, JAMES G, JETMORE
O'TOOLE, JAMES K, NEWTON
OBANDO, GUILLERMO, SALINA
OBOURN, ROBERT L, TOPEKA
OCHSNER, BRUCE B, WICHITA
ODENHEIMER, BURTRAM J, WICHITA
ODGERS, RODNEY K, PITTSBURG
*ODULIO, PERLITA, WICHITA
OELSCHLAGER, RONALD D, LAWRENCE
OHMAN, RICHARD J, DODGE CITY
OHMART, RICHARD V, OAKLEY
OKTAWIEC, DANUTA, SHAWNEE MISSION
OLD, JERRY L, ARKANSAS CITY
OLOFIELD, RAY W, KANSAS CITY
*OLIVER JR, JAMES H, DERBY
OLNEY, ROBERT D, MANHATTAN
OLSEN, PHILLIP S, EL DORADO
OLSON, CARL E, LAWRENCE
OLSON, CLITUS W, GOODLAND
OLSON, ERWIN T, NEWTON
OLTMAN, THEODORE V, RILEY
OPENSHAW, CALVIN R, HUTCHINSON
ORCHARD, RICHARD A, LAWRENCE
OSBORN DD, ROBERT M, INDEPENDENCE
OSBORNE, CONRAD C, WICHITA
OSGOOD, GEORGE M, SHAWNEE MISSION
OSIO, ANTONIO L, WICHITA
OSOBA, WILLIAM G, WICHITA
OTHMER, EKKEHARD, KANSAS CITY
OUANO JR, BIBIANO B, MC PHERSON
OVERHOLSER, NORMAN H, EL DORADO
OWEN, GARRY D, LAWRENCE
OWEN, LARUE W, WICHITA
OWEN, PERE A, WICHITA
OXLER JR, JOHN EDWARD, KANSAS CITY

P

PACE, JOHN D, PARSONS
PADGETT, WILLIAM L, WICHITA
PAGE, O VALE, PLAINVILLE
PAGE, RUTH, WICHITA
PAI, RADHA V, PARSONS
PAI, VARADARAJ S, PARSONS
PAINE, GEORGE E, HUTCHINSON
PALMBERG, KENT E, TOPEKA
PALMER, DAVID L, WICHITA
PALMER, GERALD K, SALINA
PALMER, MARVIN M, KANSAS CITY
PALMER JR, H C, LIBERAL
PANICHABHONGSE, SAMBHUNDH, RUSSELL
PAPP, ROBERT, TOPEKA
PARAMESH, JAYA, LARNED
PARANJOTHI, SUBRAMONIAM P, PARSONS
PARDO, LILLIAN G, KANSAS CITY
PARDO, MANUEL P, KANSAS CITY
PAREKH, AJITKUMAR M, KANSAS CITY
PARHAM, VERDON W, CHANUTE
PARK, CHAN H, KANSAS CITY
PARKER, HAROLD L, WICHITA
PARKER, ROBERT W, LEAVENWORTH
PARKER, WAYNE G R, EMPORIA
PARKS, GILBERT R, TOPEKA
PARMAN, ROBERT O, TOPEKA
PARRA, MIGUEL D, KANSAS CITY
PARSI, MANUTCHEHR K, PITTSBURG
PARULKAR, DEEPAK S, TOPEKA
PASCHINO, ANTONIO R, KANSAS CITY
PASIMIO, ROGER S, COLUMBUS
PASTOR, VICTOR HUGO, EMPORIA
PATAK, RAMACHANDRA V, KANSAS CITY
PATEL, VINOD, TOPEKA
PATINO, EDGAR, TOPEKA
PATRICK, FRED EDWARD, TOPEKA
PATTERSON, BRUCE W, WICHITA
PATTERSON, JOHN R, SHAWNEE MISSION
PATTERSON, MICHAEL S, GARDEN CITY
PAULS, DANIEL N, PARSONS
*PAY, NORMAN T, WICHITA
PAYNE, J RALPH, KANSAS CITY, MO
PAYNE, ROBERT R, TOPEKA
PAZELL, JOHN A, KANSAS CITY
PEARCE, EUGENE W J, SHAWNEE MISSION
PEARCE, LUNETTA M, SHAWNEE MISSION
PEASE, GARY L, HUTCHINSON
PECANA, MANUEL C, KANSAS CITY
PEDERSON, ARNOLD M, PLAINVILLE
PEDRAZA, HERNANDO V, NEWTON
PEES, GERALD B, IOLA
PEES JR, GERALD BOYD, LAWRENCE
PEFFLY, ELMER D, CHETOPA
PENCE, CHARLES O, WICHITA
PENKA, WAYNE E, TOPEKA
PENN, GEORGE M, TOPEKA
PENNINGTON, KATHERINE, WICHITA
PENTECOST, RICHARD L, SHAWNEE MISSION
PEREIRA, WILLY G, ARKANSAS CITY
PERICO, CARLOS J, SHAWNEE MISSION
PERIDO, ODMINAOOR T, ELKHART
PERKINS, JACK L, HUTCHINSON
PERKINS, WILLIAM GENE, KANSAS CITY
PERRY JR, LAWRENCE L, KANSAS CITY
PETELIN, JOSEPH B, KANSAS CITY
PETERIE, JERRY P, WICHITA
PETERS, DALE W, DALLAS, TX
PETERS, GLENN R, KANSAS CITY
PETERSEN, A GENE, SHAWNEE MISSION
PETERSEN, GERALD D, SHAWNEE MISSION
PETERSON, DEAN L, TOPEKA
PETERSON, JACK T, MANHATTAN
PETERSON, ROBERT L, TOPEKA
PETERSON, VERNON J, TOPEKA
PETERSON JR, EVAN A, WATHENA
PETIT, CARL ALFONSO, SHAWNEE MISSION
PETRIE, SAMUEL C, SHAWNEE MISSION
PETRIK, EDWIN L, TOPEKA
PETTEGREW, PAULINE K, SHAWNEE MISSION
PETTERSON, CECIL E, SYRACUSE
PETTERSON, DENNIS CRAIG, TOPEKA
PETTERSON, O'RUTH S, WICHITA
PETTIJOHN, WALTER J, RUSSELL
PFUETZE, BRUCE L, SHAWNEE MISSION
PFUETZE, KARL D, SHAWNEE MISSION
PFUETZE, ROBERT E, TOPEKA
PHAM, TOAI KE, MONTEZUMA
PHELPS, DAVID WAYNE, FORT SCOTT
PHILGREEN, DONALD E, OTTAWA
*PHILIPP, JOSEPH THEODORE, MANHATTAN
PHILLIPS, STEPHEN B, MANHATTAN
PHILLIPS, WARREN G, SHAWNEE MISSION
PHIPPS, JACK G, WICHITA
PIERCE, CHARLES F, TOPEKA
PIERCE, DONALD R, TOPEKA
PIERCE, GEORGE E, KANSAS CITY
PIERRON, GEORGE J, OLATHE
PIERRON, ROBERT L, WICHITA
PIERSON, W WEIR, MCPHERSON
PILCHARD, WILLIAM A, SHAWNEE MISSION
PINCOMB, ARTHUR L, OLATHE
PINSKER, JACOB A, WICHITA
PISCHKE, FRANK J, KANSAS CITY
PITMAN, WILL D, PRATT
PITTS, RONALD L, SHAWNEE MISSION
PLOWMAN, CARL W, JEWELL
POGSON, GEORGE W, PITTSBURG
POKORNY, CHARLES, SUN CITY, AZ
POL, P ALBERT, KANSAS CITY
POLING, TERRY L, WICHITA
POLLACK, SIMON, WICHITA
POLLAND DD, STEPHEN M, WICHITA
POLLOCK, ANTHONY G A, WICHITA
POLLY, RICHARD E, TOPEKA
POLSON, ROBERT C, GREAT BEND
POOLE, BERNARD T, WICHITA
PORTER, DAVID M, KANSAS CITY
PORTER, GARRY L, WICHITA
PORTER, ROBERT D, TOPEKA
POTTER, ROBERT L, KANSAS CITY
POWELL, CAROL W, SHAWNEE MISSION
POWELL, KENNETH A, SHAWNEE MISSION
POWELL, WILLIAM R, TOPEKA
POWELL II, BENSON M, TOPEKA
POWERS, G ROBERT, KANSAS CITY
POWERS, HAROLD W, SUN CITY, AZ
POWERS, K DEAN, WICHITA
PRAEGER, MARK A, LAWRENCE
PRAKALAPAKORN, DARANEE, NESS CITY
PRAKALAPAKORN, YANYONG, NESS CITY
PREHEIM, DELBERT V, NEWTON
PREMSINGH, NALINI G, KANSAS CITY
PRENTISS, HAROLD, HALSTEAD
PRESTON, DAVID F, KANSAS CITY
PRESTON, RALPH R, TOPEKA
PRESTON, RICHARD, GREAT BEND
PRETZ, JAMES B, KANSAS CITY
PRICE, JAMES GORDON, KANSAS CITY
PRICE, VAUGHAN C, MCPHERSON
PRICE JR, LAURANCE W, LAWRENCE
PRIETO, JORGE N, KANSAS CITY, MO
PRIETO, LUIS E, WICHITA
PROCHAZKA, OTTO F, LIBERAL
PROCTOR, ROBERT W, EL DORADO
PROKOP, BRADFORD S, TOPEKA
PRONKO, MICHAEL J, SHAWNEE MISSION
PROUD, G ONEIL, KANSAS CITY
PRUITT, JOHN C, WICHITA
PUGH, DAVID M, KANSAS CITY

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PULLMAN, NORMAN K, WICHITA
PURINTON, LEW W, WICHITA
PUTHENPURACKAL, GEORGE, WINFIELD
PUTNAM, LYLE B, WICHITA
PYLE, LUCIEN R, TOPEKA

Q

QUACKENBUSH, ROBERT P, ST JOHN
QUAISON, EMMELINE P, KANSAS CITY
QUAMAR, YUSUF, NEWTON
QUENZER, RONALD W, PRATT
QUINN, CHARLES E, KANSAS CITY
QUINONES, ELADIO A, OSAWATOMIE

R

RABE, MELVIN A, LEAVENWORTH
RACELA JR, ANTONIO S, SHAWNEE MISSION
RADOM, SANFORD B, SHAWNEE MISSION
RADOVANOV, RADMILA, NEWTON
RAHMAN, HAFIZ M A, TAMPA,FL
RAINBOW-EARHART, KATHRYN A, TOPEKA
RAMIREZ, AUGUSTO H, PITTSBURG
RAMIREZ, IRENE, PITTSBURG
RAMSEY, BARTLETT W, TOPEKA
RANDALL, GEORGE R, WICHITA
RANDLES, MICHAEL J, WICHITA
RANSDELL, EDGAR C, TOPEKA
RANSOM, JAMES H, TOPEKA
RASSA, REZA P, SALINA
RATHBUN, EDWIN D, LIBERAL
RAUSA JR, FRANCISCO C, WICHITA
RAWCLIFFE JR, ROBERT A, WICHITA
RAZEK, ZACK A, WICHITA
READ, WILLIAM T, COFFEYVILLE
REALS, WILLIAM J, WICHITA
REAZIN, WALTER L, WICHITA
RECKLING, FREDERICK W, KANSAS CITY
REDDY, B N, HILL CITY
REDDY, EASHWER K, KANSAS CITY
REDDY, P JAGANNADHA, HILL CITY
REDDY, SATTI S, DODGE CITY
REDDY, VENUMBAKA C, EL DORADO
REDFORD, JOHN W B, KANSAS CITY
REEB, RONALD JOSEPH, KANSAS CITY
REECE, A THOMEN, GARDNER
REECE, CAROL A, GARDNER
REECE, RICHARD J, SALINA
REED, A J, WICHITA
REED, D CRAMER, WICHITA
REED, DAVID D, WICHITA
REED, JAMES S, APO MIAMI,FL
REED, RALPH R, LAWRENCE
REESE, JACK D, LIBERAL
REESE, JOHN L, LAWRENCE
REEVES, BRADFORD F, WICHITA
REEVES, CHARLES S, FORT SCOTT
REGIER, HENRY L, KANSAS CITY
REGIER, LADONNA M, COLBY
REIMER, DONALD R, TOPEKA
REINHARDT-WULF, TAISSIA L, GARDEN PLAIN
REINKING, VICTOR E, TOPEKA
•REISMAN, MICHAEL ALAN, WICHITA
REITZ, LELAND C, MANHATTAN
REITZ, ROGER P, MANHATTAN
REIVICH, RONALD S, SHAWNEE MISSION
RELIHAN, DONALD A, WICHITA
RELIHAN, FRANCIS H, SMITH CENTER
REMPEL, JOHN H, WICHITA
RENDON, HUMBERTO M, AL AIN,
REPLOGLE, CHARLES B, GREAT BEND
REYES JR, FRANCISCO A, OTTAWA
REYMOND, RALPH D, TOPEKA
REYNOLDS, JEFFREY C, HAYS
REYNOLDS, LLOYD W, HAYS
RHOADS, JAMES P, WICHITA
RHODEN, CURTIS H, WICHITA
RHODES, IVAN E, WICHITA
RHODES, JAMES B, KANSAS CITY
RHODES, LOWELL M, WICHITA
RHODES, MARTIN L, KANSAS CITY
RICE JR, FREDERICK A, KANSAS CITY
RICH, ELDON S, NEWTON
RICHARDS, DALLAS LEE, HAYS
RICHARDS, DENNIS D, CLAY CENTER
RICHARDS, JON F, PHILLIPSBURG
RICHARDSON, J M, TOPEKA
RICHARDSON, JAY L, ARKANSAS CITY
RICHARDSON, STEWART F, WICHITA
RICK JR, GREGORY GP, KANSAS CITY
RIDER, JAMES W, ATCHISON
RIEDEL, ROBERT H, TOPEKA
•RIEDERER, ROBERT E, WICHITA
RIEGER, ERNEST H, WICHITA
RIEKE, FRANK A, SHAWNEE MISSION
•RIEPE, ROGER E, WICHITA
RIGLER, WILSON F, ARMA
RILEY, RAY B, KANSAS CITY
RIORDAN, HUGH D, WICHITA
RISING, JESSE D, KANSAS CITY
RIZZA, ROBERT G, HALSTEAD
ROACH, NEIL E, HALSTEAD
ROBB, JOHN B, CIMARRON
ROBERTS, DANIEL K, WICHITA
ROBERTS, LOUIS S, WICHITA
ROBERTS, RICHARD S, LAWRENCE
ROBERTS, WARREN E, TOPEKA
ROBERTSON, JOSEPH K, WICHITA
ROBINSON, DAVID B, TOPEKA
ROBINSON, DAVID W, KANSAS CITY
ROBINSON, EDGAR L, INDEPENDENCE
ROBINSON, G DONALD, WICHITA
ROBINSON, JOHN D, KANSAS CITY
ROBINSON, JOHN E, WICHITA
ROBINSON, RALPH G, KANSAS CITY
ROBINSON, ROBERT H, WICHITA
ROBISON, JAMES T, KANSAS CITY
ROBL, DAVID A, WICHITA
ROCERETO, PAUL V, TOPEKA
ROCHANAYON, PIRA, ELLIS
RODERICK, JAMES E, SALINA
RODRIGUEZ, PAUL L, GARDEN CITY
RODRIGUEZ, RAUL G, KANSAS CITY
RODRIGUEZTOCKER, LILIA, WICHITA
ROEDER, ROBERT E, TOPEKA
ROJAN, CHAVALIT, PARSONS
ROLLINS, DOUGLAS E, KANSAS CITY,MO
ROMALIS, BRIAN E, WICHITA
ROMEISER, REX S, SALINA
ROMONDO, STEVEN A, OLATHE
RONK, LEE E, KANSAS CITY
ROSABAUGH, DONALD C, ABILENE
ROSE, DONALD L, BELLA VISTA,AR
ROSE, GRAHAM C, MANHATTAN
ROSE, SHELBY D, WICHITA
ROSEN, DAVID, WICHITA
ROSENBERG, STANTON L, SHAWNEE MISSION
ROSENBERG, THOMAS F, WICHITA
ROSENTHAL, RICHARD, SHAWNEE MISSION
ROSENTHAL, STANTON J, KANSAS CITY
ROSS, DAVID K, ARKANSAS CITY
•ROSS, DENNIS LEE, WICHITA
ROSS, JACK L, TOPEKA
ROSSITTO, ANTHONY F, SAN FRANCISCO,CA
ROTERT, LARRY, TOPEKA
ROTH, ALAN E, KANSAS CITY
ROTHSTEIN, TERRY B, PARSONS
ROWLETT, JACK G, PAOLA
ROY, WILLIAM R, TOPEKA
RUBIN, HERBERT M, SHAWNEE MISSION
RUBIN JR, BEN, KANSAS CITY
RUBLE JR, JAMES L, OVERBROOK
RUCKER, CLEMENS, TOPEKA
RUEB, ANDREW E, SALINA
RUHLEN, JAMES L, OLATHE
RUIZ, CARLOS M, GREAT BEND
RUMOLD, MERVIN J, SHAWNEE MISSION
RUNNELS, JOHN B, TOPEKA
RUPP, RICHARD J, TOPEKA
RUSSELL, PHILIP W, WICHITA
RUTH, WILLIAM E, KANSAS CITY
RUZICKA, LAWRENCE J, CONCORDIA
RYAN, MICHAEL J, KANSAS CITY
RYAN, W SCOTT, EMPORIA

S

SABIN JR, GEORGE M, WICHITA
SADID, SULEMAN, WICHITA
SAEED, MOHAMMAD A, WICHITA
SAFFO, KARL S, SHAWNEE MISSION
SALGADO, CARLOS R, WICHITA
SAMUEL, CHANDY C, WINFIELD
SANCHEZ, ROGELIO, TOPEKA
SANDBERG, CHRIS B, EL DORADO
•SANDERS, GLORIA DEANNA, WICHITA
SANDERS, J ALAN, LAWRENCE
SANDHU, PAUL S, COFFEYVILLE
SANTOSCOY, GILBERT S, WICHITA
SARGENT, JOSEPH D, TOPEKA
SATHYANARAYANA, SARASWATHI, SHAWNEE MISSION
SAUL, F WILLIAM, EMPORIA
SAUNDERS, MICHAEL E, CONCORDIA
SAVIN, VIRGINIA J, KANSAS CITY
SAWKAR, LAXMIDAS A, SHAWNEE MISSION
SAYLER, JEROME, GREAT BEND
SAYLOR, EDWARD H, TOPEKA
SAYLOR, LESLIE L, TOPEKA
SAYLOR, MARK, TOPEKA
SAYLOR, STEPHEN, OTTAWA
SCALES, WILLIAM M, BLUE EYE,MO
SCAMMAN, W MIKE, TOPEKA
SCANLAN, TIMOTHY M, WICHITA
SCANLON JR, JAMES H, HADDAM,CT
SCHAFFER, JOSEPH PETER, SHAWNEE MISSION
SCHAFFER, CLARENCE K, SANTA CRUZ,CA
SCHELLINGER, RICHARD P, EMPORIA
SCHERER, ALFRED L, ST JOHN
SCHILTZ, FRANCES, WICHITA
SCHIMKE, R NEIL, KANSAS CITY
SCHLACHTER, ERNEST R, WICHITA
SCHLEMMER, ROGER B, PITTSBURG
SCHLICHER, JOHN E, WICHITA
SCHLOESSER, HARVEY L, TOPEKA
•SCHLOESSER, PATRICIA T, TOPEKA
SCHLOTTERBACK, WILLIAM E, MANKATO
SCHLUETER, JOHN J, WICHITA
SCHMAUS, LYLE F, IOLA
SCHMIDT, HERBERT R, NEWTON
SCHMIDT, RAMON WARNER, SALINA
•SCHNELLE, JOACHIM, WICHITA
SCHNOEBELEN, RENE E, KINSLEY
SCHNOSE, GREGORY D, LAWRENCE
SCHOPF, CLIFTON C, WICHITA
SCHOTLAND, EDWARD S, KANSAS CITY
SCHRAM, PETER CHARLES, TOPEKA
SCHREPFER, ROSEMARY, KANSAS CITY
SCHROEDER, SYDNEY O, LAWRENCE
SCHROLL, JACK C, HUTCHINSON
SCHUETZ, PERRY N, GREAT BEND
SCHUKMAN, JAY STEPHEN, GREAT BEND
SCHULTZ, JAMES E, COUNCIL GROVE
SCHWARTING, J STEVE, ABILENE
SCHWARTZ, EUGENE W, DODGE CITY
SCHWARTZ, V DEAN, WICHITA
SCHWEGLER, RAYMOND A, LAWRENCE
SCHWEGLER, RAYMOND A, KANSAS CITY
SCHWORM, CURTIS P, KANSAS CITY
SCLAR, WILLIAM C, SHAWNEE MISSION
SCOTT, ALEX, JUNCTION CITY
SCOTT, CHESTER E, SALINA
SCOTT, DUANE L, BELLEVILLE
SCOTT, VINCENT L, WICHITA
SCOTT, WILLIAM H, WICHITA
SEAGO, CHARLOTTE L, LIBERAL
SEAMAN, LAUREN I, OLATHE
SEBREE, STEVEN G, SALINA
SEELEY, JAMES C, HOLTON
SEGERSON, JOHN A, TOPEKA
SEGLIE, FLOYD RONALD, PITTSBURG
SEHDEV, JOAN, TOPEKA
SEITZ JR, JOSEPH E, ELLSWORTH
SEKAVEC, GORDON B, OAKLEY
SERERES, EDGAR P, KANSAS CITY
SERRANO, JOAQUIN, ARCADIA,FL
SETTLE JR, RUSSELL D, OLATHE
SETTLE SR, RUSSELL D, TOPEKA
SEVIER, SAMUEL M, TOPEKA
SHAAD, DOROTHY J, SHAWNEE MISSION
SHAFER, PRESTON J, WICHITA
SHAH, ASHOK H, WINFIELD
SHAH, MIAN, LARNED
•SHAH, MUKHTAR H, WICHITA
SHAH, NASREEN, LARNED
SHAH, SHARFUDDIN, HALSTEAD
SHAW, JAMES W, WICHITA
SHAW, JOSEPH L, TOPEKA
SHAW, RICHARD C, WICHITA
SHAW JR, JAMES W, HUTCHINSON
SHEAFOR, DOUGLAS, TOPEKA
SHEARS, ROBERT N, HUTCHINSON
SHEERN, MARK DOUGLAS, ABILENE
SHEFFER, KEITH D, OLATHE
SHEIKH, MASOOD A, INDEPENDENCE
SHELLITO, JOHN G, WICHITA
SHELTON, STEPHEN E, TOPEKA
SHEPARD, LEROY W, LARNED
SHEPPARD, ROBERT G, SMITH CENTER
SHERMAN, ROBERT P, KANSAS CITY
SHERWOOD JR, CLARENCE E, TOPEKA
SHIELDS JR, JAMES M, EL DORADO
SHIFLET, ALBERT W, WICHITA
SHIVEL, DAVID G, GREAT BEND
SHOFSTALL, WILLIAM H, SHAWNEE MISSION
SHORTES, LOIS E, OSWEGO
SHRADER, DOYLE A, WICHITA
SHUSS, JOHN LOGAN, KANSAS CITY
SIBALA, JUSTO L, PRATT
SIEGEL, ALBERT R, WICHITA
SIEGEL, CARL D, WICHITA
SIEMENS, RICHARD A, LYONS
SIFERS, EARL C, KANSAS CITY
SIFERS, TIMOTHY M, KANSAS CITY
SIFFORD, R LAWRENCE, WICHITA
SILER, EUGENE T, HAYS
SILLS, CHARLES T, NEWTON
SILVERS, ALVIN, KANSAS CITY
SIMMONS, ROBERT EARLE, NEWTON
SIMPSON, J COLBERT, SALINA
SIMPSON, ROBERT LIMBAUGH, OBERLIN
SIMPSON, TOM C, STERLING
SIMPSON, WILLIAM S, TOPEKA
SINGER, PHILIP A, KANSAS CITY,MO
SINGH, GIRVAR, ARKANSAS CITY
SINNING, GARY, HIAWATHA
SISK, PHILLIP B, TOPEKA
SIWEK, CHRISTOPHER W, EL DORADO
SKAER, STANLEY ALLEN, EUREKA
SKIBBA, RICHARD M, WICHITA
SKIKNE, BARRY S, KANSAS CITY
SLEEPER, CAROL A, KANSAS CITY,MO
SLEEPER, DONALD C, ESPANOLA,NM
SLOD, MILO G, SALINA
•SLUTSKY, LAWRENCE JOEL, WICHITA
SMITH, ALVIN L, WICHITA
SMITH, BOYD E, SALINA
SMITH, BRUCE G, ARKANSAS CITY
SMITH, DALE C, SHAWNEE MISSION
SMITH, DONALD J, SHAWNEE MISSION
SMITH, HAROLD R, SALINA
SMITH, JOHN D, LARNED
SMITH, LED A, TOPEKA
SMITH, NEWTON C, ARKANSAS CITY
•SMITH, STEPHEN BUHLER, TOPEKA
SMITH, STEPHEN D, KANSAS CITY
SMITH, STEPHEN J, ARKANSAS CITY
•SMITH, TIMOTHY WM, WICHITA
SMITH JR, FLOYD L, COLBY
SMITH JR, WILLARD J, WICHITA
SNARR, JACK W, TOPEKA
SNODELL, FIRMIN E, SHAWNEE MISSION
SNODK, ROBERT RUFUS, MCLOUTH
SNOW, DONALD L, LANSING
SNOW JR, ARTHUR D, SHAWNEE MISSION
SNOWBARGER, MARVIN D, EMPORIA
SNYDER, C JOHN, WINFIELD

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SNYDER, GREGG M, WICHITA
SNYDER, HOWARD E, WINFIELD
SNYDER, THOMAS E, MONTGOMERY,AL
SNYDER JR, RICHARD HENRY, OLATHE
SOELDNER, JAMES OLIVER, SHAWNEE MISSION
SOHLBERG JR, ROBERT, MCPHERSON
SOLOMON, HERMAN, WICHITA
SOLTZ, ROBERT A, WICHITA
SOMERS, MARVIN M, WICHITA
SONGER, HERBERT L, LINCOLN
SOUCEK, CHARLES O, KANSAS CITY
SPANN, RICHARD W, WICHITA
SPAULDING, JOHN S, KANSAS CITY
SPEARMAN, JESSE L, TOPEKA
SPEER, FREDERIC, SHAWNEE MISSION
SPEER, LELAND, KANSAS CITY
SPEER, LOUIS N, OTTAWA
SPENCER, JOHN HAROLD, FORT SCOTT
SPENCER, MILLARD C, TOPEKA
SPENCER, WAYNE E, TOPEKA
SPIKES, MARION E, GARDEN CITY
STAFFORD, ROBERT W, HUTCHINSON
STANLEY, KENNETH E, WICHITA
STANLEY, REX C, PAOLA
STARK, JAMES R, WICHITA
STARKEY, JERALD L, RUSSELL
STECH, JOSEPH M, ANDALE
STECHSCHULTE, DANIEL J, KANSAS CITY
*STECKLEY, RICHARD ALLEN, WICHITA
STEEGMANN, A THEODORE, INDIANAPOLIS,IN
STEELE, CLARENCE H, KANSAS CITY
STEICHEN, EDWARD F, LENORA
STEIN, JOSEPH M, TOPEKA
STEIN, PAUL S, WICHITA
STEIN, ROBERT B, MANHATTAN
STEIN, ROBERT THOMAS, KANSAS CITY,MO
STEINER, ROBERT M, SHAWNEE MISSION
STEINKRUGER, VERLYN WILLIAM, SMITH CENTER
STEINZEIG, ALFRED S, SHAWNEE MISSION
STEINZEIG, SHERMAN M, KANSAS CITY
STENSAAS, CARL D, HUTCHINSON
STEPHENS, CHARLES, MINNEOLA
STEPHENS, RONALD L, KANSAS CITY
STEPHENSON, LUCILLE C, ST FRANCIS
STEVENS, MILDRED J, GARNETT
STEVENS, PHILIP L, TONGANOXIE
STEVENS, ROBERT L, GARNETT
STEVENSON, CHARLES E, KANSAS CITY
STEVENSON, E KENT, SHAWNEE MISSION
STEWART, DAVID R, KANSAS CITY
STEWART, JACK T, WICHITA
STILLIE, DO, G DONALD, KANSAS CITY,MO
STITT, RONALD W, SHAWNEE MISSION
STOCK, KARL W, TOPEKA
STOCKWELL, MORGAN U, DODGE CITY
STOFER, BERT E, PRESCOTT,AZ
STOFFER, ROBERT P, HALSTEAD
STOKES, ROBERT LEE, KANSAS CITY,MO
STOLZ, ELMER G, WICHITA
STONE, G REX, MANHATTAN
STONE, GRANT C, ATTICA
STOSKOPF, LAWRENCE E, WICHITA
STOUT, JAMES M, HUTCHINSON
STOUT, NILES M, LYNDON
STRAHM, WAYMER J, INDEPENDENCE
STREET, DAVID E, WICHITA
STREHLOW, CHESTER H, OTTAWA
*STRICKLAND, MAURICE H VAN, WICHITA
STRUTZ, WILLIAM C, LEAVENWORTH
STRYKER, TERRY MARGARET, WICHITA
STRYKER JR, HENRY B, CONCORDIA
STUBBLEFIELD, CHARLES T, KANSAS CITY
STUBER, JACK LAWRENCE, SHAWNEE MISSION
STUCKY, DEAN E, MEDICINE LODGE
STUMP, HARL G, HAYS
SUERO, JESUS T, WICHITA
SUFI, MOHAMMAD ASHRAF, TOPEKA
SUFI, DAISER A, TOPEKA
SUGAR, ROBERT L, SHAWNEE MISSION
SUITER, DANIEL JAY, PRATT
SULLIVAN, CORNELIUS J P, WICHITA
SULLIVAN, LEONARD L, WICHITA
SULLIVAN, TOMMY GRAY, KANSAS CITY
SULLIVAN JR, HENRY B, SHAWNEE MISSION
SUMNER, JOYCE R, HUTCHINSON
SUMNER, MARION M, HUTCHINSON
SUMNER, RALPH N, FREDONIA
SURFACE, GARDNER A, ELLIS
SUTTON, RICHARD O, TOPEKA
SUTTON, ROBERT E, INDEPENDENCE
SUTTON JR, RICHARD L, SHAWNEE MISSION
SVOBODA, CHARLES R, CHAPMAN
SWAN, MAJOR MARTIN, GREAT BEND
SWANN, CLAIR L, RUSSELL
*SWANSON, HOWARD J, WICHITA
SWARTZ, WARREN E, PARSONS
SWEENEY, JAMES G, WICHITA
SWISHER, WILLIAM C, WICHITA
SWOGGER JR, GLENN, TOPEKA

T

*TADURAN, VIRGILIO, SATANTA
TAMBURINI, MARIO, LEAVENWORTH
TANDOC JR, VALENTIN T, NEWTON
TANGCHUPONG, CHANTRASIRI, PARSONS
TANGCHUPONG, SAROHD, PARSONS
TAPPEN, DANIEL L, TOPEKA
TARGOWNIK, KARL K, TOPEKA
TARNOFF, GERALD M, TOPEKA
TARNOWER, WILLIAM, TOPEKA
*TATPATI, DANIEL A, WICHITA
*TATPATI, OLGA ADELINA, WICHITA
TAYIEM, ABDEL K, ATCHISON
TAYLOR, ELMER W, SEDAN
TAYLOR, RICHARD J, WICHITA
TAYLOR, STEVEN L, WICHITA
TAYLOR, THOMAS F, SALINA
TAYLOR, THOMAS L, KANSAS CITY
TEARE, MAX E, GARDEN CITY
TEJANO, NEONILO A, HALSTEAD
TEMPERO, STEPHEN J, TOPEKA
TEMPLETON, ARCH W, KANSAS CITY
TETZLAFF, ARCHIBALD O A, SHAWNEE MISSION
THELEN, J CHRISTINE, WICHITA
THEROU, LEONA F, KANSAS CITY
THIEMANN, A H, D.O., SUBLETTE
THOMAS, GREGORY MCQUEEN, MCPHERSON
THOMAS, JAMES H, KANSAS CITY
THOMAS, R CULLEN, PRATT
THOMAS, THOMAS V, KANSAS CITY
THOMPSON, DANIEL M, WICHITA
THOMPSON, DANNIE M, KANSAS CITY
THOMS, NORMAN W, TOPEKA
THOMSEN, GARY, SHAWNEE MISSION
THORNTON JR, FOXHALL P, CONCORDIA
THORPE, FRANCIS A, PRATT
THURSTON, DAVID E, TOPEKA
TIEMANN, WILLIAM H, MANHATTAN
TIHEN, EDWARD N, WICHITA
TIHEN, HENRY N, WICHITA
TILLER, GEORGE R, WICHITA
TILLOTSON, DON R, ULYSSES
TILTON, FRANK M, WICHITA
TINKER, ROBERT C, WICHITA
TINTEROW, MAURICE M, WICHITA
TIPPIN JR, ERNEST E, WICHITA
TIVORSAK, ARKOM, ATCHISON
TOALSON, WILLIAM B, KANSAS CITY
TOCKER, ALFRED M, WICHITA
TOMPKINS, CARL O, NEWTON
TONN, GERHART R, WICHITA
TOTH, JOHN ROY, TOPEKA
TOZER, RICHARD C, TOPEKA
TRACY, TERRY A, WICHITA
TRAVIS, JOHN W, TOPEKA
TREES, CLYDE B, TOPEKA
TREES, DONALD P, WICHITA
TREGER, NEWMAN V, TOPEKA
TREKELL, WILLIAM V, DODGE CITY
TRETBAR, HARVEY A, WICHITA
TRETBAR, LAWRENCE L, SHAWNEE MISSION
TREWEEKE, MICHAEL W, WICHITA
TRIMBLE SR, DAVID P, EMPORIA
TROPP DO, ARNOLD L, SHAWNEE MISSION
TROTTER, ROGER COURTNEY, MINNEOLA
*TRUDEAU, DAVID L, WICHITA
TRUEWORTHY, ROBERT C, KANSAS CITY
TRUITT, WILLIAM ROY, TOPEKA
TUCKER, DONALD R, LAWRENCE
TUCKER, SHERIDAN G, DE RIDDER,LA
TUCKER, VIRGINIA L, LAWRENCE
TUONG, TRAN MANH, AUGUSTA
TURNER, JOHN W, GARDEN CITY
TUTERA, GINO, SHAWNEE MISSION
TWEET, FREDRICK A, PITTSBURG
TWEITO, DAVID H, HUTCHINSON
TWEMLOW, STUART W, TOPEKA
TYLER, WILLIAM F, FT GORDEN,GA

U

UBELAKER, ERNEST J, SOUTH HAVEN
UHLIG, PAUL J, WICHITA
UHR, NATHANIEL, TOPEKA
UNDERWOOD, CHARLES C, EMPORIA
UNREIN, ROBERT J, GREAT BEND
UTLEY, JAMES HARMON, KANSAS CITY
UY, WILSON O, COFFEYVILLE

V

VACHAL, EVA, GARDEN CITY
VAKAS, JOHN L, COFFEYVILLE
VALK, WILLIAM L, SHAWNEE MISSION
VAN DER VLIET, STEPHEN K, WICHITA
VAN HOUDEN, CHAS E, WICHITA
VAN THULLENAR, PHILIP A, KANSAS CITY
VANDE GARDE, LARRY O, TOPEKA
VANDER VELDE, STANLEY L, EMPORIA
VANNAMAN, DONALD D, SHAWNEE MISSION
VARGHESE, GEORGE, KANSAS CITY
VATS, TRIBHAWAN S, KANSAS CITY
VAUGHAN, DONNA A, NEWTON
VELARDE, HUGO, SHAWNEE MISSION
VEMIREDDI, NANDA KUMAR, KANSAS CITY,MO
VERMA, ASHA, PARSONS
VILLANUEVA, CESAR L, KANSAS CITY
VILLARANTE, FE TEJERO, KANSAS CITY
VIN ZANT, LARRY E, WICHITA
*VINE, DONALD LEE, WICHITA
*VINZANT, MARK N, DERBY
VINZANT, WHITNEY L, WICHITA
VISSER, VALYA E, KANSAS CITY
VISWANATHAN, BYRAVAN, TOPEKA
VOGEL, STANLEY J, TOPEKA
VOGT, VERNON W, NEWTON
VOLKMANN II, HARLEY W, MANHATTAN
VON GKASEMSIRI, SUNAN, NEWTON
VON LEONROD JR, GEORGE, DIGHTON
VON RUDEN, WILLIAM J, HUTCHINSON
VOORHEES, CARROLL O, LEAVENWORTH
VOORHEES, GORDON S, LEAVENWORTH
VOPAT, RICHARD L, WICHITA
VORHEES, VICTOR J, WICHITA
VOTAPKA, WILLIAM L, STOCKTON
VOTH, DOUGLAS W, WICHITA
VOTH, HAROLD M, TOPEKA
VRANEY, GEORGE, HALSTEAD

W

WADDELL, BILL O, KANSAS CITY
WADE, THEODORE E, LIBERAL
WADUD, ABDUL, WICHITA
WAGENBLAST, HOWARD R, SALINA
WAGGONER, FRANKLIN E, BONNER SPRINGS
WALDORF JR, MELVIN H, GREENSBURG
WALKER, JACK D, KANSAS CITY
WALKER, MAURICE A, KANSAS CITY
WALKER, NELLIE G, LEES SUMMIT,MO
WALKER, WILLIAM H, ESKRIDGE
WALKER, WILLIAM K, SEDAN
WALKER, WILLIAM L, SHAWNEE MISSION
WALLACE, LEO F, TOPEKA
WALLACE, ROBERT W, SEDONA,AZ
WALLACE JR, WAYNE O, ATCHISON
WALLS, WILLIAM J, TOPEKA
WALSH, RICHARD A, WICHITA
WALSH, THOMAS E, ONAGA
WALTERS, BYRON W, LAWRENCE
*WALTERS, ROBERT MERRILL, WICHITA
WALTON, PHILIP D, ATWOOD
WALZ, ROYCE C, TOPEKA
WALZ, THOMAS J, ST FRANCIS
WANG, SIDNEY W, SHAWNEE MISSION
WARD, HOWARD N, TOPEKA
WARD, JAMES A, BELLEVILLE
WARD, ROBERT L, STAFFORD
WARE, LUCILE M, TOPEKA
WARREN, LINDA D, HANOVER
WARREN, LLOYD P, WICHITA
WARREN, ROGER D, HANOVER
WARREN, WIRT A, WICHITA
WARREN JR, JOHN W, WICHITA
WARRICK, DAVID ALAN, TOPEKA
WATERS, CLARENCE N, SALINA
*WATERS, DALE A, TOPEKA
WATERS III, CHESTER H, OMAHA,NE
WATTS, HARRY E, HAYS
WAXMAN, DAVID, KANSAS CITY
WAYLAN, THORNTON L, NASHVILLE
WEARE, MARY E, SHAWNEE MISSION
WEAVER, J ROBERT, WICHITA
WEAVER, JACK D, WICHITA
WEAVER, WALTER D, TOPEKA
WEBB, JAMES R, SHAWNEE MISSION
WEBER, CLARENCE J, SALINA
WEBER, DARRELL J, TOPEKA
WEBER, ROBERT W, SALINA
WEBER, ROY R, NEWTON
WEBER, WALLACE N, HAYS
WEBER II, RALPH H, SALINA
WEBER JR, HUGO P, WICHITA
WEBSTER, BOBBY W, WICHITA
WEDDLE, DOUGLAS P, FORT SCOTT
WEDEL, KENNETH D, MINNEAPOLIS
WEDEL, KERMIT G, MINNEAPOLIS
WEDIN, PAUL H, WICHITA
WEIGAND, JOEL T, WELLINGTON
WEIGEL, JOHN W, KANSAS CITY
WEIPPERT, EDWARD J, WICHITA
WEIR, WILLIAM C, ERIE
WELCH, JACK W, HALSTEAD
WELCH, LAUREN A, GARDEN CITY
WELCH, LAUREN K, WICHITA
WELCH, MARTIN H, WICHITA
WELCH, MAURA S, GARDEN CITY
WELL, MICHAEL A, LAWRENCE
WELLER, ELIZABETH B, KANSAS CITY
WELLER, RONALD ALAN, KANSAS CITY
WELLS, ALVIN Y, WINFIELD
WELLS, BRUCE W, WINFIELD
*WELLS, MAX MICHAEL, WICHITA
WELLSHEAR, CHARLES C, WICHITA
WELTMER, ROGER P, BELOIT
WENINGER, JOHN H, WICHITA
WENZEL, ANNA MARIE, HAYS
WERNER, WILLARD F, TRIBUNE
WERTH, CLAUDE J, SHAWNEE MISSION
WERTZBERGER, JOHN, LAWRENCE
WERTZBERGER, KENNETH LYNN, LAWRENCE
WESBROOK, CLYDE W, N Y,NY
WESCOE, W CLARKE, NEW YORK,NY
WEST, C OMER, KANSAS CITY
WEST, WILLIAM T, WICHITA
WHALLON, JACOB T, WICHITA
WHEELER, DWIGHT E, NEWTON
*WHEELER, NICKY RAY, WICHITA
WHEELER, PINCKNEY R, WICHITA
WHITAKER, JAMES A, WICHITA
WHITAKER, REN R, OBERLIN
WHITE, CHARLES L, GREAT BEND
WHITE, CHARLES M, WICHITA

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WHITE, DONALD C, COFFEYVILLE
WHITE, FAGAN N, RUSSELL
WHITE, HARRY H, TOPEKA
*WHITE, JOHN D, WICHITA
WHITE, JOHN P, PARSONS
WHITE, R BURNLEY, WINFIELD
WHITE, THADDEUS H, MANHATTAN
WHITE II, BENJAMIN E, EL DORADO
WHITEHEAD, RICHARD E, SHAWNEE MISSION
WHITLEY, DOUGLAS M, SHAWNEE MISSION
*WIBERG, THOMAS A, WICHITA
WIEDERMAN, FRANCIS JAMES, TOPEKA
WIEGMAN, HUGH ALAN, HAYS
WIENS, J WENDELL, NEWTON
WIENS, PETER K, NESS CITY
WIGGINTON, GERALD D, D.O., SHAWNEE MISSION
WIGGLESWORTH, ANNE, TOPEKA
WIGGS, JAMES W, GREAT BEND
WIKSTEN, VERNON C, TOPEKA
WILCHINS, LAWRENCE J, LAS VEGAS,NV
WILCOX, DONALD E, TOPEKA
WILCOX JR, HOWARD L, HAYS
WILCOX SR, HOWARD L, LAWRENCE
WILDER, LOWELL W, WICHITA
WILDS, CHARLES E, TOPEKA
WILEY, HORACE M, GARDEN CITY
WILEY, JOHN H, SHAWNEE MISSION
*WILKINSON, LARRY K, WICHITA
WILLIAMS, CHARLES L, WICHITA
WILLIAMS, EVAN ROBERT, DODGE CITY
WILLIAMS, HOMER J, OSAGE CITY
WILLIAMS, HOWARD V, WICHITA
WILLIAMS JR, STERLING B, KANSAS CITY
WILLIAMSON, JOHN, HUTCHINSON
WILLIAMSON, JOHN A, KANSAS CITY
WILSON, COL JAMES W, FT RILEY
WILSON, H RANDOLPH, HALSTEAD
WILSON, L BARRICK, SHAWNEE MISSION
WILSON, MARVIN H, TOPEKA
WILSON, ROBERT B, SHAWNEE MISSION
WILSON, ROBERT L, VALLEY CENTER
WILSON, SLOAN J, SHAWNEE MISSION
WINBLAD, JAMES N, WINFIELD
WINCHELL, H H FORSYTH, WICHITA
WINCHESTER, EUGENE B, WICHITA
WING, NANCY J, TOPEKA
WINN, RANDALL S, KANSAS CITY
WINTER JR, CALVERT J, KANSAS CITY
WISDOM, JAY K, WICHITA
WISE III, JOSEPH EDWARD, KANSAS CITY
WISNER JR, HARRY J, WICHITA
WISNER JR, JOHN HENRY, KANSAS CITY
WITTMANN, ALBERT F, WICHITA
WOLF, CURTIS V, LYONS
WOLF, KARL T, KANSAS CITY
WOLE, PATRICK G, WICHITA
WOLFE, FREDERICK, WICHITA
WOLFE, FREDERICK P, PRATT
WOLKOFF, CDR A STARK, SAN FRANCISCO, CA
WOLLMANN, MARTIN, LAWRENCE
WONG, BERT Y, KANSAS CITY
WONG, NORMUND, TOPEKA
WOOD, DOUGLAS H, PITTSBURG
*WOOD, EDWARD R, TOPEKA
WOOD, GARY B, WICHITA
WOOD, ROBERT D, HORTON
WOODARD, LAWRENCE E, WICHITA
WOODHOUSE, CHARLES L, WICHITA
WOODS, HUGH J, SMITH CENTER
WOODS, ROBERT P, TOPEKA
WOOLLCOTT JR, PHILIP, CHICAGO,IL
WORTMAN, JACK A, HUTCHINSON
WRAY, ALEXANDER J, WICHITA
WRIGHT, KENDALL M, EMPORIA
WRIGHT, M ERIK, LAWRENCE
WRIGHT, STANLEY E, JUNCTION CITY
WRIGHT JR, ROBERT W, KANSAS CITY
*WU, JIN-TZE, WICHITA
WULFF, EDWIN T, ATCHISON
WURSTER, GEORGE R, SHAWNEE MISSION

Y

YAGHMOUR, TALAAT E, PITTSBURG
YE, RICHARD C, SHAWNEE MISSION
YEOMANS, RONALD N, OLATHE
YODER, EMERSON D, DENTON
YODER, VERNON E, HESSTON
YOHE, RUTH M, SHAWNEE MISSION
YOUMANS, ROGER L, AFRICA,
YOUNG, CHARLES H, ATCHISON
*YOUNG, DOUGLAS L, WICHITA
YOUNG, JOHN W, SHAWNEE MISSION
YOUNG, THEODORE E, TOPEKA
YOUNGBERG, DEAN I, WICHITA
YOUNGLOVE, R HAL, SHAWNEE MISSION
YOUNGSTROM, KARL A, SHAWNEE MISSION
YULICH, JOHN D, KANSAS CITY

Z

ZABEL, KENNETH P, PITTSBURG
ZACHARIAS, CARL KURT, DODGE CITY
ZACHARIAS, DAVID LLOYD, TOPEKA
ZACK, ASHLEY S, SHAWNEE MISSION
ZAMIEROWSKI, DAVID S, SHAWNEE MISSION
ZAREMSKI, SHERMAN C, SHAWNEE MISSION
ZARNOW, HILARY, WICHITA
*ZATZKIN, JAY B, WICHITA
ZELLER, MYRON J, GARDEN CITY
ZIEGLER, DEWEY K, KANSAS CITY
ZIMMERMAN, DANIEL D, SHAWNEE MISSION
ZIMMERMAN, KENNETH D, WICHITA
ZIMMERMAN, WILLIAM H, TOPEKA
ZINN, THOMAS W, KANSAS CITY
ZONGKER, PHILIP E, WICHITA

* Probationary members.

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Medical Specialty Codes Used in This Directory

A	Allergy
ADM	Administrative Medicine
ADT	Addictionology
AM	Aviation Medicine
ANES	Anesthesiology
CD	Cardiovascular Disease
CDS	Cardiovascular Surgery
CDTS	Cardiovascular & Thoracic Surgery
CHN	Child Neurology
CHP	Child Psychiatry
CP	Clinical Pharmacology
D	Dermatology
DR	Diagnostic Roentgenology
EENT	Eye, Ear, Nose and Throat
EM	Emergency Medicine
END	Endocrinology
ENT	Ear, Nose, Throat
FM	Family Medicine
FP	Family Practice
GE	Gastroenterology
GEN	Genetics
GER	Geriatrics
GP	General Practice
GPM	General Preventive Medicine
GS	General Surgery
GYN	Gynecology
HEM	Hematology
ID	Infectious Diseases
IE	Insurance Examination
IM	Internal Medicine
MDST	Medical Student
MO	Medical Oncology
N	Neurology
NEP	Nephrology
NM	Nuclear Medicine
NP	Neuropsychiatry
NR	Nuclear Radiology
NS	Neurological Surgery
OBG	Obstetrics and Gynecology
OM	Occupational Medicine
ON	Oncology
OPH	Ophthalmology
ORS	Orthopedic Surgery
OS	Other Specialties
OST	Osteopathy
OTO	Otorhinolaryngology
P	Psychiatry
PATH	Pathology
PD	Pediatrics
PDA	Pediatric Allergy
PDC	Pediatric Cardiology
PDN	Pediatric Neurology
PDO	Pediatric Ophthalmology
PDR	Pediatric Radiology
PDS	Pediatric Surgery
PED	Pediatric Cardiology
PH	Public Health
PM	Physical Medicine & Rehabilitation
PNP	Pediatric Nephrology
PS	Plastic Surgery
PUD	Pulmonary Disease
R	Radiology
RES	Resident
RHU	Rheumatology
RM	Rehabilitative Medicine
RO	Radiology/Oncology
RT	Radiation Therapy
SON	Surgical Oncology
TR	Therapeutic Radiology
TS	Thoracic Surgery
U	Urology
00	Retired

Physician Distribution by Cities

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Line 1: Doe, John R., 1234 Oak St., 67052
(Name) (Street Address) (Zip Code)

Line 2: (654-2222)
(Telephone Number)

Line 3: 33 M 1902 58 FP
(Year of Birth) (Sex) (Medical School) (Year of Graduation) (Specialty)

Telephone area code follows city name. * Probationary Members

ABILENE—913
(Dickinson County Society)

BERKLEY, DON H, 1111 N BRADY,67410
263-4131
35 M 1902 61 FP
BIGGS, DENNIS, 1405 N CEDAR,67410
263-7190
48 M 1902 74 FP
BUTCHER, ARCHIE W, 303 HILLTOP DR,67410
-
01 M 1902 35 OO
CHAFFEE, DEAN C, RR 1,67410
263-4131
11 M 1902 44 FP
COLEMAN, GARY, 1405 N CEDAR,67410
-
M FP
HEINS, LAWRENCE G, RR #5,67410
-
97 M 4102 21 OO
MOHLER, JACK M, RR 4,67410
263-4131
32 M 1902 61 FP
NARCISO, VICENTE O, 515 NE 10TH ST,67410
263-2253
45 M 74810 68 GS
RORABAUGH, DONALD C, PROFESSIONAL BLDG,67410
263-4131
33 M 1902 58 FP
SCHWARTING, J STEVE, 1405 N CEDAR,67410
263-7190
46 M 3401 72 FP
SHEERN, MARK DOUGLAS, 1111 N BRADY,67410
263-4131
51 M 1902 76 FP

ALMA—913
(Pottawatomie County Society)

MCKNIGHT, ELLIS B, ,66401
765-3317
03 M 1902 33 FP

ALTAMONT—316
(Labette County Society)

JACKSON, VICTOR L, BOX 467,67330
784-5318
20 M 2105 50 FP

ANDALE—316
(Sedgewick County Society)

STECH, JOSEPH M, ANDALE CLINIC,67001
445-2152
27 M 3006 56 FP

ANTHONY—316
(Tri-County Society)

ANTRIM, PHILIP JENIFER, 1101 E SPRING,67003
842-5144
15 M 1902 42 FP
HALL, JERRY D, 1101 E SPRING,67003
842-5144
31 M 3901 56 FP

ARKANSAS CITY—316
(Cowley County Society)

ALVAREZ, NORBERTO, 112 E CENTRAL,67005
442-4850
27 M 27501 53 FP
AUCAR, ALFREDO, BOX 1105,67005
442-1710
23 M 27501 53 OTO
CAMPBELL, GARLAND L, 114 W WALNUT,67005
442-1350
13 M 1902 40 FP
CHAVALA, SUDARSAN, 2508 EDGEMONT DR,67005
442-4300
43 M 49511 68 OPH
FARRAR JR, WILLIAM H, 2508 EDGEMONT,67005
442-8200
44 M 1902 70 IM
GREEN, LAWRENCE C, 112 EAST CENTRAL AV,67005
442-4850
43 M 3901 69 FP
HINSHAW, EDGAR D, RT #3,67005
442-1273
15 M 1902 51 R
LEIKER, JOSEPH, 201 A C OFFICE BLDG,67005
442-7900
48 M 1902 73 IM
MEEK, GEORGE C, PO BOX 1148,67005
442-2100
05 M 1902 32 FP
OLO, JERRY L, PO BOX 1148,67005
442-2100
49 M 1902 74 FP
PEREIRA, WILLY G, 2508 EDGEMONT DR,67005
442-8540
39 M 73701 67 IM
RICHARDSON, JAY L, 2508 EDGEMONT DRIVE,67005
442-4150
38 M 1902 65 GS
ROSS, DAVID K, PO BOX 1148,67005
442-2100
48 M 1902 75 FP
SINGH, GIRVAR, 2508 EDGEMONT DR,67005
442-4300
40 M 49555 64 OPH
SMITH, BRUCE G, 115 E RADIO LANE,67005
442-5600
20 M 1902 44 IM
SMITH, NEWTON C, 510 W RADIO LANE,67005
442-2100
21 M 3901 45 FP
SMITH, STEPHEN J, 115 HILLSIDE,67005
442-5600
45 M 1902 71 FP

ARMA—316
(Crawford County Society)

RIGLER, WILSON F, 511 WASHINGTON,66712
347-8619
42 M 1803 69 FP

ASHLAND—316
(Iroquois County Society)

MC NICKLE, GEORGE ANDREW, BOX 907,67831
635-2222
47 M 1902 75 FP

ATCHISON—913
(Atchison County Society)

BOSSE, FRANK K, 1301 RIVERVIEW DRIVE,66002
-
09 M 2802 33 OO
BRADY, CHARLES S, 111 N 5TH ST,66002
367-1232
11 M 3006 38 GS
BRIBACH, EUGENE J, 125 1/2 N 5TH,66002
367-0225
83 M 2802 05 OPH

BROWN, ROBERT O, 1400 NORTH 2ND,66002
367-1922
14 M 1902 44 FP
BURKE, JOSEPH V, 1301 N 3RD,66002
367-5496
35 M 3006 66 GS
FAST, ROBERT E, 122S N 2ND,66002
367-0362
48 M 1902 74 DBG
FAST, W SPENCER, RAMSAY MED CLINIC,66002
367-0362
11 M 3006 39 FP
GROWNEY, JOHN T, 801 ATCHISON,66002
367-5020
37 M 3006 63 FP
HART, LAWRENCE E, 1412 N 2ND,66002
367-S0S4
32 M 1902 64 FP
MORRISON, IRA R, 825 N 10TH,66002
367-4396
07 M 1611 36 1M
RIDER, JAMES W, 122S N 2ND ST,66002
367-0362
47 M 2803 73 FP
TAYIEM, ABDEL K, 122S N 2ND,66002
367-0362
43 M 33002 68 GS
TIVORSAK, ARKOM, 1716 COUNTRY LANE,66002
367-2131
40 M 89101 68 R
WALLACE JR, WAYNE O, 1301 N THIRD,66002
367-7300
36 M 2803 6S FP
WULFF, EDWIN T, 111 N STH,66002
367-5033
07 M 2834 36 FP
YOUNG, CHARLES H, 1301 N 3RD,66002
367-40S3
23 M 1902 S3 FP

ATTICA—316
(Tri-County Society)

STONE, GRANT C, 215 W AVE O,67009
2S4-7219
08 M 5605 35 FP

ATWOOD—316
(Northwest Kansas Society)

DILL, ROONEY SIDNEY, 411 PAGE,67730
626-3229
41 M 77 GP
WALTON, PHILIP O, S14 MAIN,67730
626-3322
32 M 1902 63 FP

AUGUSTA—316
(Butler-Greenwood Society)

ANDERSON, DALE W, 209 D WEST 7TH STREET,67010
77S-S432
30 M 1902 S5 FP
BARBER, JAMES L, AUGUSTA PLAZA,67010
77S-S432
31 M 1902 S7 FP
LAHAM, ALEXANDER J, RR 3 BOX 87,67010
683-4658
20 M 1902 44 1M
TUONG, TRAN MANH, AUGUSTA PLAZA,67010
775-5432
39 M 65 GP

BAXTER SPRINGS—316
(Cherokee County Society)

ALQUIST, VERYL D, 21ST & FAIRVIEW,66713
623-4942
17 M 1902 42 GS
CHUBB, RICHARD M, 44S EAST 10TH,66713
8S6-2444
29 M 1606 S4 FP
MEHAFFY, ORVILLE A, 411 E 11TH,66713
8S6-2383
42 M 1902 69 FP
MOU, BIN, 445 E TENTH,66713
8S6-2444
23 M 24402 S0 1M

BELLEVILLE—913
(Republic County Society)

BEIDERWELL, PAUL L, 2703 M ST,66935
08 M 3901 38 FP
CHANEY, ERNIE J, 2316 G STREET,6693S
S27-2237
27 M 1902 S6 FP
DOUBEK, HERBERT D, 2316 G ST,6693S
S27-2237
28 M 1902 S6 FP
SCOTT, DUANE L, BELLEVILLE CLINIC,66935
527-2217
34 M 1902 60 FP
WARD, JAMES A, 1206 18TH ST,6693S
527-2217
34 M 1902 S8 FP

BELOIT—913
(Mitchell County Society)

DOBRATZ, ROBERT A, 310 W 8TH,67420
738-2246
24 M 1902 S2 FP
DRAKE, DOUGLAS J, MEDICAL CENTER,67420
738-2246
43 M 1902 71 FP
KLENDA JR, MARTIN B, BELOIT MED CENTER,67420
738-2246
38 M 1643 63 GS
LONEY, JOHN M, 310 WEST 8TH,67420
738-2246
SO M 1902 74 1M
WELTMER, ROGER P, 112 W MAIN,67420
738-2S74
18 M 1902 44 U

BLUE RAPIDS—913
(Northeast Kansas Society)

LAWLESS, HAROLD L, 607 LINCOLN,66411
226-7202
29 M 702 54 FP

BONNER SPRINGS—913
(Wyandotte County Society)

MAY, KENNETH L, 122 N NETTLETON,66012
422-2020
20 M 1902 S1 FP
MITTS, ERNEST W, 122 N NETTLETON,66012
422-2020
22 M 1902 S1 FP
WAGGONER, FRANKLIN E, 122 N NETTLETON,66012
422-2020
26 M 1902 61 FP

BUCKLIN—316
(Ford County Society)

BETTERTON, DALE C, PO BOX 607,67834
826-32S1
38 M 2604 73 FP

BUHLER—316
(Reno County Society)

FRIESEN, ORLANDO J, 107 W 2ND,67S22
S43-2330
27 M 1902 S6 FP

CALDWELL—316
(Tri-County Society)

KINNAN, L F, S23 S MARKET,67022
84S-6422
18 M 3901 42 FP

CANEY—316
(Southeast Kansas Society)

MOORE, ROBERT F, 4TH & MCGEE,67333
879-2135
28 M 1902 S6 FP

CEDAR VALE—316
(Southeast Kansas Society)

COHNBERG, ROSELLEN E, PO BOX 399,67024
7S8-2266
22 F 2802 47 FP

CHANUTE—316
(Southeast Kansas Society)

ABBUEHL, DON R, S05 SOUTH PLUMMER,66720
431-2S00
18 M 1902 44 GS
ASHLEY, SAMUEL G, 505 SOUTH PLUMMER,66720
431-2S00
16 M 1902 43 FP

BAKER, HENRY K, 811 WEST MAIN,66720
431-1600
08 M 1606 35 GS
BURKMAN, REUBEN J, 1501 W 7TH,66720
431-9310
28 M 1902 54 FP
DICK JR, HENRY J, 1501 W 7TH,66720
431-9310
27 M 1902 58 FP
GEHRT, EARL B, 505 SO PLUMMER,66720
431-2500
32 M 1902 62 FP
HASKINS, ROBERT J, 505 S PLUMMER,66720
431-2500
46 M 1902 74 FP
HUDSON, JAMES R, 616 HILLSIDE,66720
431-4000
37 M 1902 63 R
KIHM, ALBERT A, 505 S PLUMMER,66720
431-2500
27 M 1902 55 FP
MIH, ALEXANDER, 1002 WEST 4TH,66720
473-2227
22 M 24209 47 ANES
PARHAM, VERDON W, 505 S PLUMMER,67349
431-2500
47 M 1902 73 FP

CHAPMAN—316
(Dickinson County Society)

SVOBODA, CHARLES R, 413 N MARSHALL,67431
922-6400
18 M 1902 46 FP

CHENEY—316
(Sedgwick County Society)

MEHRHOF, EDWARD G, 219 W FIRST,67205
542-3543
32 M 3515 62 FP

CHERRYVALE—316
(Labette County Society)

MCKENZIE, STEVE LEWIS, 216 E FOURTH,67335
336-2131
47 M 2878 77 GP

CHETOPA—316
(Labette County Society)

PEFFLY, ELMER O, 327 MAPLE,67336
236-7188
22 M 3901 53 FP

CIMARRON—316
(Ford County Society)

ROBB, JOHN B, FAMILY PRACTICE CLINIC,67835
855-2251
36 M 80301 60 FP

CLAY CENTER—913
(Clay County Society)

ANDERSON, SEVERT A, 1749 BERGLUND DR,67432
-
07 M 1902 36 OD
CARLETON, RICHARD C, 709 LIBERTY,67432
532-5603
31 M 3005 61 FP
DALUM, PETER JOSEPH, 709 LIBERTY SUITE C,67432
632-2181
45 M 2803 76 FP
MCVAY, R BRUCE, 1503 FIFTH ST,67432
-
97 M 1902 29 OD
O'DONNELL, RICHARD H, 709 LIBERTY,67432
632-3101
16 M 1902 41 GS
RICHARDS, DENNIS O, 115 S 6TH,67432
632-5621
34 M 1902 60 FP

CLYDE—913
(Cloud County Society)

FREEBORN JR, WARREN S, ,66938
446-2221
26 M 1720 51 FP

COFFEYVILLE—316
(Southeast Kansas Society)

BRYANT, HOMER L, 803 W 9TH,67337
259-3890
99 M 3501 30 OPH
CAMPBELL, WILLIAM H, 1411 W 4TH,67337
251-3235
39 M 1902 65 OPH
COYLE, JOHN F, 209 W 7TH,67337
251-2400
21 M 1902 44 FP
DICKINSON, CHARLES R, 108 W 7TH,67337
251-1340
20 M 1606 44 GS
DIXON, RAYMOND W, 1411 W 4TH,67337
251-1090
M GS
EOROZO, M LUZ LUNA, PO BOX 856,67337
251-1200
F 74801 68 PATH
ELLIS, STEPHEN S, 1411 W 4TH,67337
251-3360
11 M 2802 36 GS
GIBBS, EUGENE, PO BOX 716,67337
251-7260
M 64914 68 FP
HA, SANG W, 504 WILSHIRE,67337
251-7750
35 M 58309 60 OBG
HAN, CHAN S, 908 SIGGINS,67337
251-1560
35 M 58306 61 PD
HOWERTER JR, BERNARD E, PO BOX 659,67337
251-4790
43 M 1803 68 U
READ, WILLIAM T, 1411 WEST 4TH,67337
251-1120
16 M 2802 40 FP
SANDHU, PAUL S, PO BOX 257,67337
251-2450
42 M 49509 65 GS
UY, WILSON O, COFFEYVILLE MEM HOSPITAL,67337
251-1200
42 M 74801 67 PATH
VAKAS, JOHN L, 1508 W 4TH,67337
251-3443
38 M 1902 64 IM
WHITE, DONALD C, PO BOX 262,67337
251-1200
35 M 3515 65 R

COLBY—913
(Northwest Kansas Society)

DAHL, ASHER W, COLBY CLINIC,67701
462-3333
28 M 1902 58 FP
HASSETT, GERARD R, 187S HARVEY,67701
-
24 M 3006 50 R
HILOYARD, VICTOR H, COLBY CLINIC,67701
462-3333
16 M 1902 42 OTO
HILOYARD II, VICTOR H, BOX 28,67701
462-3332
47 M 702 73 FP
JACOBSEN, DWIGHT SKINNER, COLBY CLINIC,67701
462-3333
31 M 3545 60 GS
MARSHALL, GEORGE D, COLBY CLINIC,67701
462-3333
09 M 1902 36 FP
REGIER, LADONNA M, COLBY CLINIC,67701
462-3332
47 F 1902 73 FP
SMITH JR, FLOYD L, COLBY CLINIC,67701
462-3333
20 M 1902 44 FP

COLDWATER—316
(Iroquois County Society)

GOERING, DONALD D, BOX 576,67029
582-2136
31 M 1902 56 FP

COLUMBUS—316
(Cherokee County Society)

BELCHER, GEORGE D, BOX 309,66725
429-2557
34 M 1902 60 FP
JONES, FORREST H, 219 S KANSAS,66725
429-3744
25 M 1902 54 FP
PASIMIO, ROGER S, R2 BOX 259,66725
429-1977
38 M 74801 62 GS

COLWICH—316
(Sedgwick County Society)

LIES, BARTHEL N, 309 S THIRD,67030
796-1255
11 M 2802 37 FP

CONCORDIA—913
(Cloud County Society)

BRAY, AVIS PAGE, 1010 3RD AVE,66901
243-1560
17 F 702 54 FP
BUTT, MUHAMMAD, CONCORDIA MED GROUP,66901
243-1560
46 M 70401 69 GS
FOWLER, WAYNE L, CONCORDIA MED GROUP,66901
423-1560
23 M 1720 47 IM
GELVIN, E RAYMOND, 835 WEST 9TH,66901
243-1560
03 M 3005 27 GS
HOFER, DEWAYNE O, COUNTRY CLUB RD,66901
243-1263
36 M 1606 62 R
KOSAR, CLARENCE D, BOX 362,66901
98 M 1902 26 OO
LAWTON, MARVIN K, CONCORDIA MED GROUP,66901
243-1560
31 M 3005 58 GS
LLOYD, JAMES W, 910 WEST 11TH,66901
243-7011
44 M 1902 73 FP
MCCOMAS JR, MARMADUKE D, 11TH & WASHINGTON ST,66901
243-2511
16 M 1902 43 U
MOORE, JAMES E, 910 W ELEVENTH,66901
243-1094
48 M 1902 74 P
NELSON, PAUL L, 1010 THIRD AVE,66901
243-1560
27 M 1902 55 PD
NIXON, RICHARD R, 519 WASHINGTON,66901
243-1263
32 M 1643 57 R
RUZICKA, LAWRENCE J, 1010 3RD AVE,66901
243-1560
13 M 3005 40 ANES
SAUNDERS, MICHAEL E, 1404 CRESCENT LANE,66901
243-1560
50 M 1902 73 IM
STRYKER JR, HENRY B, 717 FIRST AVE,66901
19 M 3501 44 OO
THORNTON JR, FOXHALL P, 1010 3RD AVE,66901
243-1560
25 M 5101 50 IM

COTTONWOOD FALLS—316
(Flint Hills Society)

MCKEE, LEO F, ,66845
273-6681
16 M 1902 39 FP

COUNCIL GROVE—316
(Flint Hills Society)

BARKER, ROYAL A, 221 HOCKADAY,66846
767-5126
21 M 1902 53 FP
BLACKBURN, ROBERT W, 221 HOCKADAY,66846
767-5126
22 M 1902 49 FP
SCHULTZ, JAMES E, 221 HOCKADAY,66846
767-5126
26 M 1902 56 FP

CUNNINGHAM—316
(Wyandotte Society)

ALLBRITTEN JR, FRANK F, PO BOX 177,67035
14 M 4101 38 OO

DENTON—913
(Northeast Kansas Society)

YODER, EMERSON O, ,66017
359-6531
14 M 1902 49 FP

DERBY—316
(Sedgwick County Society)

MCKERRACHER, ROBERT O, 400 A NORTH BALTIMORE,67037
688-1779
27 M 3901 55 FP
MILLER, LAWRENCE H, 1410 N WOODLAWN,67037
788-3741
40 M 1001 67 FP
MOYER, HERMAN J, 200 S BALTIMORE,67037
788-1484
24 M 3901 55 FP
OLIVER JR, JAMES H, 229 S GEORGIE,67037
788-2867
30 M 1606 57 AM
VINZANT, MARK N, 1410 N WOODLAWN,67037
681-3092
45 M 64914 75 FP

DIGHTON—316
(Southwest Kansas Society)

VON LEONROD JR, GEORGE, PO BOX 146,67839
397-5314
16 M 1902 43 FP

DODGE CITY—316
(Ford County Society)

AMAWI, MOHAMMAD S, DODGE CITY MED CENTER,67801
225-1371
46 M 87501 71 GS
AVILA, OSCAR A, DODGE CITY MED CENTER,67801
225-1371
41 M 17603 69 IM
AYUTHIA, ISSARA I, 3001 AVENUE A,67801
40 M 89101 66 PATH
BAUM, ARNOLD H, DODGE CITY MED CENTER,67801
225-1371
16 M 2101 44 OBG
BOLES, R DALE, DODGE CITY MED CENTER,67801
225-1371
23 M 3901 53 PD
BROWNRIGG, RICHARD L, DODGE CITY MED CENTER,67801
225-1371
35 M 1902 61 U
BUSCH, ANTHONY B, SOUTHWEST CLINIC,67801
05 M 2002 31 OO
CHOTIMONGKOL, ANUPONG, DODGE CITY MED CENTER,67801
225-1371
43 M 89102 69 OBG
CONARD, CLAIR C, DODGE CITY MED CENTER,67801
225-1371
27 M 1902 55 IM
GREENBERG, GEORGE E, 1904 BURR PARKWAY,67801
276-8241
42 M 401 68 R
JAMBOR, JAMES J, 304 MCARTOR,67801
227-3213
20 M 2307 46 O
JOHNSON, HOWELL O, DODGE CITY MED CENTER,67801
225-1371
45 M 1902 71 IM
KUNNAWUTHIDEE, KAMOLTIPYA, 2020 CENTRAL, 67801
225-1371
45 F 89101 61 PD
MCCOY, RONALD, SOUTHWEST CLINIC,67801
227-3141
19 M 3901 47 FP
MCELHINNEY, CHARLES F, DODGE CITY MED CENTER,67801
227-1371
36 M 1902 62 GS
MCGINNIS, MICHAEL J, SOUTHWEST CLINIC,67801
227-3141
41 M 5501 69 GS
MIMOSO, JOSE J, SOUTHEWST CLINIC,67801
227-3141
37 M 4201 61 OBG
OHMAN, RICHARD J, 1810 1/2 FAIRWAY DR,67801
15 M 2407 41 GS
REDDY, SATTI S, DODGE CITY MED CENTER,67801
225-1371
35 M 49504 66 U
SCHWARTZ, EUGENE W, 1ST NATL BANK BLDG,67801
225-4261
24 M 1902 50 OPH
STOCKWELL, MORGAN U, 1904 BURR PARKWAY,67801
225-1371
24 M 1902 55 IM
TREKELL, WILLIAM V, SOUTHWEST CLINIC,67801
227-3141
25 M 1902 52 ORS

WILLIAMS, EVAN ROBERT, DODGE CITY MED CENTER,67801
225-1371
25 M 1605 52 FP
ZACHARIAS, CARL KURT, DODGE CITY MED CENTER,67801
225-1371
21 M 40707 47 ORS

EL DORADO—316
(Butler-Greenwood Society)

BRIAN, ROBERT M, 123 N ATCHISON,67042
321-1230
02 M 1606 30 FP
DELLETT, KENNETH B, 3RD & VINE,67042
321-1910
30 M 1902 55 OPH
GIROD, CHARLES I, 123 N ATCHISON ST #103,67042
321-4981
11 M 4706 44 GS
HAFFNER, WILLIAM N, 123 N ATCHISON,67042
321-5049
35 M 1902 61 GS
JACOB, KANNAMPALLY L, 123 N ATCHISON,67042
321-0056
31 M 49537 59 U
KASSEBAUM, GLEN E, 123 N ATCHISON,67042
321-5082
98 M 1606 23 GS
LEE, YONG U, 123 N ATCHISON,67042
321-5630
35 M 60 GS
OLSEN, PHILLIP S, 123 N ATCHISON,67042
321-2100
46 M 1902 73 IM
OVERHOLSER, NORMAN H, 119 N JONES,67042
321-2010
16 M 1902 41 FP
PROCTOR, ROBERT W, 119 N JONES,67042
321-2010
38 M 1902 63 FP
REDDY, VENUMBAKA C, ROOM 302,67042
321-3300
46 M 49511 70 IM
SANDBERG, CHRIS B, 119 NORTH JONES,67042
321-2010
48 M 1902 74 FP
SHIELDS JR, JAMES M, 119 N JONES,67042
321-2010
18 M 4802 42 FP
SIWEK, CHRISTOPHER W, 123 N ATCHISON,67042
321-5211
48 M 75911 71 ORS
WHITE II, BENJAMIN E, 119 N JONES,67042
321-2010
27 M 1902 54 FP

ELKHART—316
(Seward County Society)

IWAY, BELINO D, PO BOX 460,67950
697-2175
42 M 74811 66 IM
PERIDO, DOMINADOR T, BOX 997,67950
697-2155
44 M 74801 68 GS

ELLINWOOD—316
(Barton County Society)

LAW, FINDLEY, MEDICAL ARTS BLDG,67526
564-2170
22 M 1902 51 FP
MORRISON, RICHARD L, 611 N MAIN,67526
564-2423
42 M 1902 67 FP

ELLIS—913
(Central Kansas Society)

ROCHANAYON, PIRA, 814 JEFFERSON ST,67637
726-4956
43 M 89101 69 FP
SURFACE, GARDNER A, 1204 WASHINGTON ST,67637
-
02 M 1902 29 DO

ELLSWORTH—913
(Central Kansas Society)

DAVIS, GEORGE R, 308 KINGSLEY,67439
472-3121
19 M 1902 44 GS
O'DONNELL, HAROLD F, 412 BLAKE,67439
-
99 M 1902 26 OD
SEITZ JR, JOSEPH E, 308 KINGSLEY,67439
472-3121
22 M 1902 46 FP

EMPORIA—316
(Flint Hills Society)

BEST, JOHN F, 919 W 12TH, 66801
342-2521
51 M 2803 77 IM
BRADLEY, H RUSSELL, 1601 STATE,66801
343-2900
35 M 1902 61 FP
BROCKHOUSE, JOHN P, 1601 STATE,66801
343-2900
31 M 1902 57 IM
BURGESON, FRANK G, 1601 STATE,66801
342-6989
40 M 3005 65 OPH
BUTCHER, THOMAS P, 1128 LAWRENCE,66801
342-0722
05 M 1601 34 GS
CAMPBELL, EDWARD G, 1601 STATE,66801
343-2900
31 M 1902 61 FP
COLDSMITH, DONALD C, 1024 W 12TH,66801
342-7047
26 M 1902 58 FP
COX, JACK A, 1601 STATE,66801
343-6990
36 M 702 66 ENT
DAVIS, DAVID R, 1025 STATE ST APT #2,66801
342-0646
02 M 2101 28 OO
EDWARDS, DAVID J, 1601 STATE ST,66801
343-1191
43 M 2803 69 ORS
FRAZIER, RICHARD L, 1005 W 12TH,66801
342-5137
40 M 1902 72 GS
GANN, E LAMONTE, RR #2,66801
-
07 M 2802 37 OO
GARCIA, GOULD C, 919 WEST 12TH AVE,66801
342-2521
32 M 3607 58 IM
GEITZ, JAMES M, 919 W 12TH,66801
342-2521
46 M 1902 72 IM
GINAVAN, DUANE A, 1024 W 12TH,66801
342-5876
35 M 1902 62 FP
GLENN, JAMES N, 1601 STATE,66801
343-1191
40 M 4804 66 ORS
HARVEY, JOHN E, 2506 W 15TH,66801
343-2900
39 M 1902 65 OBG
HAVENHILL II, MARSHALL A, 812 ANDERSON ST,66801
343-6400
35 M 1902 61 OBG
HOPPER, CHARLES R, 25 W 5TH,66801
342-2341
17 M 1902 47 FP
KEITH, ROBERT MARSHALL, MENTAL HEALTH CLINIC,66507
342-0548
27 M 801 54 P
KELLING, COLLYER, NEWMAN HOSP,66801
342-7722
50 M 1902 75 DR
KNECHT, STEPHEN M, NEWMAN MEMORIAL HOSP,66801
342-7722
44 M 1902 70 R
LOHMEYER, KENNETH L, 1024 WEST 12TH,66801
342-6622
18 M 1902 44 FP
LUEDTKE, WALTER E, NEWMAN MEM HOS,66801
343-6800
18 M 5605 43 PATH
MIGUELIND, OLIVER M, NEWMAN HOSP,66801
343-6800
35 M 74801 57 PATH
MORGAN, JOHN L, 919 WEST 12TH,66801
342-2521
15 M 4101 40 IM
NEIGHBOR, RALPH M, 827 COMMERCIAL ST,66801
343-6565
46 M 1902 72 OBG
NEUER, FREDERICK S, NEWMAN HOSP,66801
342-7722
46 M 3601 71 R
PARKER, WAYNE G R, EMPORIA STATE UNIVERSITY,66801
343-1200
27 M 1902 56 FP
PASTOR, VICTOR HUGO, 1601 STATE,66801
342-7715
43 M 13202 68 U

RYAN, W SCOTT, 2510 W FIFTEENTH,66801
343-7590
47 M 1902 73 PD
SAUL, F WILLIAM, ROUTE #5,66801
-
07 M 4113 40 OD
SCHELLINGER, RICHARD P, 1128 LAWRENCE,66801
342-0722
22 M 3005 49 GS
SNOWBARGER, MARVIN O, 1601 STATE STREET,66801
343-2900
29 M 1902 55 FP
TRIMBLE SR, DAVID P, 517 MERCHANT STREET,66801
342-2572
04 M 1902 32 OPH
UNDERWOOD, CHARLES C, 25 WEST 5TH,66801
342-2341
07 M 1902 32 IM
VANDER VELDE, STANLEY L, 1601 STATE,66801
343-2900
16 M 1902 43 GS
WRIGHT, KENDALL M, 1024 WEST 12TH,66801
343-2376
45 M 1902 71 FP

ERIE—316
(Southeast Kansas Society)

BRYAN, EMERY C, RT 2 BOX 171,66733
-
04 M 1902 32 OD
WEIR, WILLIAM C, 115 S MAIN,66733
-
17 M 2501 42 OD

ESKRIDGE—913
(Flint Hills Society)

WALKER, WILLIAM H, 2ND & CEDAR,66423
449-2301
10 M 2401 38 IM

EUDORA—913
(Douglas County Society)

HOLLADAY, KENNETH R, 101 WEST 10TH,66025
542-2345
34 M 1902 58 FP

EUREKA—316
(Butler-Greenwood Society)

CISKEY, WILLIAM J, 1602 NORTH ELM,67045
583-7401
47 M 1902 72 FP
SKAER, STANLEY ALLEN, 100 E 16TH,67045
583-7486
40 M 3901 65 GS

FORT RILEY—913
(Geary County Society)

WILSON, COL JAMES W, CHIEF OF PROFESSIONAL SVC,66442
862-9360
26 M 3901 58 PATH

FORT SCOTT—316
(Bourbon County Society)

AKERS, GUY I, 710 W 8TH,66701
223-3100
20 M 1902 53 FP
ALDIS, HENRY, 710 W 8TH,66701
223-3100
13 M 1902 41 OBG
ALDIS, WILLIAM, 710 WEST 8TH,66701
223-3100
20 M 1902 44 GS
BASHAM, JAMES J, 102 S JUDSON,66701
223-4100
14 M 1902 37 FP
BENAGE, JOHN F, 710 W 8TH,66701
223-3100
32 M 1902 58 OBG
BRAUN, EDWARD W, 710 WEST 8TH,66701
223-3100
42 M 1902 68 U
BURKE, JAMES J, 710 W 8TH,66701
223-3100
35 M 2834 61 IM
CHOW, STANLEY Y, 821 BURKE,66701
223-2200
18 M 24222 39 R
DUNSHEE, CARLYLE M, 710 W 8TH,66701
223-3100
32 M 1902 57 GS

GETTLER, DEAN T, 710 WEST 8TH,66701
223-3100
31 M 1902 57 GS
GOOD, JAMES T, 821 BURKE,66701
223-2200
21 M 2802 45 PATH
GRIMALDI, GARY A, 710 W EIGHTH,66701
223-3100
M OBG
IRBY, ADDISON C, 416 S JUDSON,66701
-
05 M 1606 28 OD
IRBY, PRATT, 710 WEST 8TH,66701
223-3100
13 M 4705 36 U
MCCANN, PATRICK E, 710 WEST 8TH,66701
223-3100
28 M 1902 59 IM
MCKENNA, MICHAEL J, 102 SOUTH JUDSON,66701
223-4100
38 M 1902 64 FP
NELSON, T EUGENE, 710 W 8TH,66701
223-3100
41 M 1902 69 FP
NICHOLS, ROBERT R, 102 S JUDSON,66701
223-4100
50 M 2803 76 FM
PHELPS, DAVID WAYNE, 102 S JUDSON,66701
223-4100
51 M 1902 76 FP
REEVES, CHARLES S, 710 W 8TH,66701
223-3100
37 M 1902 63 IM
SPENCER, JOHN HAROLD, 710 W 8TH,66701
223-3100
47 M 1902 74 FP
WEDDLE, DOUGLAS P, 710 WEST 8TH,66701
223-3100
43 M 1720 69 FP

FREDONIA—316
(Southeast Kansas Society)

BACANI, OSWALDO, 1324 ROBINSON,66736
378-3700
44 M 74810 70 GS
BAYLES, HUGH G, PO BOX 30,66736
378-3412
25 M 1902 52 FP
BEAL, RAYMOND J, 600 MADISON,66736
378-2159
12 M 1902 38 GS
SUMNER, RALPH N, 712 MADISON,66736
378-2311
31 M 1902 57 FP

GARDEN CITY—316
(Southwest Kansas Society)

ARTILES, BENJAMIN HIPOL, ST CATHERINE HOSP,67846
275-6111
29 M 54 P
AUSTIN, JOHN O, 601 N 6TH,67846
276-2346
14 M 1601 40 FP
BARNARD III, JAMES A, 1601 HARDING,67846
275-9671
31 M 4812 62 OBG
BEGGS, DAVID F, BOX 1077,67846
275-9671
39 M 1902 64 IM
BIGLER, F CALVIN, 2320 BELMONT PLACE,67846
275-9671
31 M 801 57 GS
BRUNO, JAMES W, 1133 KANSAS PLAZA,67846
276-8201
42 M 4706 66 FP
CALBECK, JOHN, 2603 BELMONT PLACE,67846
275-9671
50 M 1902 75 IM
EICHHORN, FRANK D, BOX 719,67846
276-8132
25 M 1902 56 FP
FENTON, ROBERT M, 710 PERSHING,67846
276-8201
20 M 1902 54 FP
FRY, LUTHER L, 603 N FIFTH,67846
275-9671
41 M 1902 67 OPH
GARDINER, TED M, 603 N FIFTH,67846
275-9671
M PD

GILBERT II, JOHN H, BOX 1077,67846
275-9671
46 M 1902 70 ORS
JENKINS, EDWARD, 1133 KANSAS PLAZA,67846
276-8201
M 506 61 FP
KALBAC, RICHARD W, BOX 1077,67846
275-9671
45 M 2803 70 OBG
KOKSAL, TOM, PLAZA MED CENTER,67846
276-8201
M FP
MAXFIELD, RUSSELL J, 1133 KANSAS PLAZA,67846
276-8201
16 M 1902 41 FP
MEYERS, STEPHEN, BOX 1077,67846
275-9671
48 M 2834 70 PD
MILLER, ROBERT E, BOX 1077,67846
275-9671
26 M 4812 55 GS
PATTERSON, MICHAEL S, BOX 1077,67846
275-9671
43 M 1902 69 OM
RODRIGUEZ, PAUL L, #306 CEDAR SUITE 101,67846
275-6111
39 M 4706 66 R
SPIKES, MARION E, 603 N 5TH,67846
275-9671
26 M 1902 62 FP
TEARE, MAX E, 1007 DAVIS,67846
276-7689
28 M 1902 54 O
TURNER, JOHN W, 210 E SPRUCE,67846
276-3292
13 M 1902 39 FP
VACHAL, EVA, 608 N FIFTH,67846
275-6111
F PATH
WELCH, LAUREN A, 603 N FIFTH,67846
45 M GS
WELCH, MAURA S, 603 N FIFTH,67846
275-9611
50 F OBG
WILEY, HORACE M, BOX 1768,67846
376-6901
12 M 2802 40 GS
ZELLER, MYRON J, BOX 1077,67846
275-9671
38 M 1902 64 OM

GARDEN PLAIN—316
(Sedgwick County Society)

BIERMANN, ALOYSIUS H, ,67050
535-2231
99 M 3005 23 FP
REINHARDT-WULF, TAISSIA L, PO BOX 273,67050
19 F 91302 42 OO

GARDNER—913
(Johnson County Society)

HALL DO, KENDALL WM, RT 1 BOX 173B,66030
884-8711
46 M 2878 76 GP
REECE, A THOMEN, 121 HICKORY CIRCLE,66030
884-8711
37 M 1902 63 FP
REECE, CAROL A, 121 HICKORY CIRCLE,66030
884-8711
37 F 1902 63 PD

GARNETT—913
(Anderson County Society)

DOUGHERTY, THOMAS M, 117 W 6TH,66032
448-5421
28 M 1902 55 FP
HARRIS JR, CLAIB B, 320 S OAK ST,66032
448-5431
17 M 1902 44 FP
LEITCH, DAVID A, GARNETT MEDICAL CENTER,66032
448-5421
38 M 1902 63 FP
STEVENS, MILDRED J, 202 W 4TH,66032
448-5454
23 F 1902 47 FP
STEVENS, ROBERT L, 202 WEST 4TH,66032
448-5454
23 M 1902 47 FP

GIRARD—316
(Crawford County Society)

FRIGGERI, ROBERT W, 111 N SUMMIT,66743
724-8723
23 M 1902 51 FP
HALL, WESLEY H, PO BOX 158, 66743
724-6154
25 M 1902 57 FP

GLASCO—913
(Cloud County Society)

HARWOOD, CLAUDE J, PO BOX 428,67445
568-2342
25 M 1902 55 FP

GOESSEL—316
(Marion County Society)

LOPEZ, RUBEN ENRIQUE, 305 E MAIN,67053
367-2261
37 M 27501 60 GP

GOODLAND—913
(Northwest Kansas Society)

AUSTIN, KENNETH D, 520 MAIN,67735
899-3633
33 M 3005 63 FP
FITZGERALD, JAMES E, 520 MAIN,67735
626-3229
44 M 77 GP
LONG, LLOYD O, 520 MAIN,67735
899-5651
37 M 1720 63 FP
MCCULLOUGH, ROBERT C, 126 E 10TH BOX 180,67735
899-3633
25 M 702 58 GP
OLSON, CLITUS W, 520 MAIN ST,67735
899-3633
16 M 3005 48 GS

GREAT BEND—316
(Barton County Society)

ALDERSON, THOMAS WHITNEY, 1400 POLK,67530
792-5341
50 M 1902 75 FP
ANDERSON, LYLE B, 1636 CHEROKEE, 67530
793-8141
28 M 1902 60 FP
BEAHM, ANOL W, 3923 BROADWAY,67530
793-7827
16 M 1902 43 FP
BEAHM, DONALD E, MED ARTS BLDG,67530
792-3626
45 M 1902 71 OPH
BROWN, C REIFF, 1031 JACKSON,67530
792-4391
31 M 3901 57 ORS
CAVANAUGH, CLAIR J, C K M C,67530
792-2617
23 M 1803 47 R
DEGNER, JAMES B, 3515 BROADWAY,67530
792-2617
31 M 1902 57 R
ELLEDGE, E FRED, 2222 FOREST,67530
792-3678
38 M 1902 65 U
EVANS, WILLIAM R, 3923 BROADWAY,67530
793-3574
25 M 1902 53 FP
FIESER, CARL W, 3515 BROADWAY,67530
792-2617
45 M 1902 71 R
GATENO, JOSEPH, 1023 JACKSON SQUARE,67530
793-3501
25 M 64901 50 OBG
HOLT, JOHN M, 2200 LAKIN,67530
793-8429
35 M 1902 61 IM
JONES, EDWARD L, 3515 BROADWAY,67530
792-2511
35 M 1902 61 PATH
KING, WILLIAM T, 3431 FOREST,67530
793-3501
35 M 1902 61 OBG
KIRBY, MERLIN G, 1012 WASHINGTON,67530
793-3091
31 M 1902 56 GS
KRUEGER, HAVEN C, 1023 JACKSON SQUARE,67530
792-2163
32 M 1902 61 PD

MCALLASTER, WENOALE E, 2111 FOREST,67530
793-3591
24 M 1902 54 GS
NIEDEREE, W CURTIS, 1012 WASHINGTON,67530
793-3091
30 M 3006 56 GS
POLSON, ROBERT C, 80X A 1422 POLK ST,67530
793-8414
17 M 1902 42 OPH
PRESTON, RICHARO, 2200 LAKIN,67530
793-8426
42 M 1902 69 IM
REPLOGLE, CHARLES 8, 2111 FOREST,67530
793-3591
27 M 1902 53 FP
RUIZ, CARLOS M, 3107 TWELFTH,67S30
792-3210
25 M 27501 52 P
SAYLER, JEROME, CENTRAL KS MEDICAL CENTER,67530
792-2S11
20 M 4113 50 PATH
SCHUETZ, PERRY N, 1422 POLK BOX A,67530
793-8414
45 M 1902 71 OPH
SCHUKMAN, JAY STEPHEN, 1400 POLK,67530
792-S341
50 M 1902 75 FP
SHIVEL, DAVID G, 3523 FOREST,67S30
793-3523
28 M 1902 55 FP
SWAN, MAJOR MARTIN, 3923 8ROADWAY,67530
792-4S40
06 M 1902 43 IM
UNREIN, ROBERT J, 1017A JACKSON,67530
792-2504
29 M 1902 58 FP
WHITE, CHARLES L, 2412 OOVE TERRACE,67530
-
06 M 1902 36 OO
WIGGS, JAMES W, 1027 JACKSON,67530
792-1336
36 M 1720 63 N

GREENSBURG—316
(Iroquois County Society)

8RADLEY, J ROOERICK, 502 SOUTH WALNUT,67054
723-2127
23 M 1902 47 FP
WALDORF JR, MELVIN H, 8RAOLEY-WALOORF CLINIC,67054
723-2127
23 M 1902 47 FP

HALSTEAD—316
(Harvey County Society)

AILLON, ALEJANORO J, HERTZLER CLINIC,67056
835-2241
39 M 26402 63 TS
BAILEY, COLIN, HERTZLER CLINIC,67056
835-2241
33 M 35205 59 GYN
8EUGELSOIJK, HENRY PETER, HERTZLER CLINIC,670S6
835-2241
49 M 1902 74 ANES
8OUOREAUX, VELTIN J, HERTZLER CLINIC,67056
835-2241
37 M 4812 64 R
8URNETT, A OEAN, HERTZLER CLINIC,67056
835-2241
21 M 1902 52 GS
DECKER, DONALO O, HERTZLER CLINIC,67056
835-2241
31 M 1902 56 CD
EASTES, GARY OEAN, HERTZLER CLINIC,670S6
835-2241
44 M 4812 71 U
GNAU, FREORIC 8, 803 MAIN,67056
835-2241
42 M 1902 68 OTO
HARMS, WILMER A, THE HERTZLER CLINIC,67056
835-2241
22 M 1902 56 OPH
HILL, PHILLIP K, THE HERTZLER CLINIC,67056
283-3600
29 M 1803 56 IM
HOLT, ROBERT G, 327 CHESTNUT,67056
83S-2241
22 M SI04 44 OPH
HOOFER, WILFORO O, HERTZLER CLINIC,67056
835-2241
30 M 1902 55 TS
MALONE, EUGENE M, HERTZLER CLINIC,67056
835-2241
23 M 1902 56 IM
MARSH, CONNIE M, HERTZLER CLINIC,67056
835-2241
47 F 1902 75 IM
MONTGOMERY, LLOYD DAN, HERTZLER CLINIC,67056
835-224I
43 M 360I 69 P
MONTGOMERYSHORT, RUTH G, HERTZLER CLINIC,67056
835-224I
I0 F I902 37 ENT
MORTON, JOHN E, HERTZLER CLINIC,67056
835-2241
99 M 35211 26 P
PRENTISS, HAROLD, 4TH AND CHESTNUT,67056
835-2241
36 M I720 62 R
RIZZA, ROBERT G, RT #2,67056
-
30 M 1201 56 PO
ROACH, NEIL E, HERTZLER CLINIC,67056
835-2241
38 M I902 67 CP
SHAH, SHARFUDOIN, HERTZLER CLINIC,67056
835-2241
3I M 7040I 58 IM
STOFFER, ROBERT P, HERTZLER CLINIC,67056
835-2241
26 M 1902 48 IM
TEJANO, NEONILO A, HERTZLER CLINIC,67056
835-2241
43 M 74808 67 ORS
VRANEY, GEORGE, HERTZLER CLINIC,67056
835-2241
43 M 5606 69 IM
WELCH, JACK W, HERTZLER CLINIC,67056
835-2241
I8 M I902 51 GS
WILSON, H RANOOLPH, HERTZLER CLINIC,67056
835-224I
20 M 4II2 45 GYN

HANOVER—913
(Northeast Kansas Society)

WARREN, LINOA O, 80X 38,66945
325-2240
44 F 1902 70 FP
WARREN, ROGER O, 80X 38,66945
337-2214
31 M 1902 57 GS

HARPER—316
(Tri-County Society)

8ELLAR, RALPH E, 1019 CENTRAL,67058
896-3131
3I M 3005 60 FP
FORREO JR, WALTER A, 121 E MAIN,67058
896-3661
43 M 1902 69 FP
GARDNER, 8ILLIE L, 121 E MAIN,67058
896-3661
25 M 1902 57 FP

HAVEN—316
(Reno County Society)

HAINES, CHESTER W, 102 SUNSET LANE,67543
-
01 M 1606 28 OO

HAYS—913
(Central Kansas Society)

ALLEN, JAMES E, 2707 VINE #6,6760I
628-3261
46 M 1902 72 IM
APPLEGATE JR, FRANCIS R, 1010 DOWNING,6760I
628-8218
30 M 1902 SS OPH
ARTMAN, JOHN C, 2005 LINCOLN ORIVE,67601
625-2518
24 M 2002 48 ANES
8ULA, RALPH E, 3205-A WILLOW,67601
-
12 M 1902 37 OO
CARLSON, EARL V, 2818 N VINE ST,67601
628-8221
31 M 300S 56 ORS
CECIL III, JOHN, 80X 833,67601
625-6521
43 M 4804 69 R
CODY, OOROTHY, 2704 WOOOROW CT,67601
628-4000
29 F 3607 53 P

CODY, JOHN, 2704 WOODROW CT,67601
628-2871
25 M 401 60 P
COX, ROBERT H, 2507 CANTERBURY RD,67601
625-2551
43 M 1902 70 PD
CRAMM, RUSSELL E, 3004 BROADWAY,67601
625-2518
30 M 1902 56 GS
EDDY, VICTOR M, 105 W 13TH,67601
625-2551
29 M 1902 55 GS
ENBERG, ROBERT, 2507 CANTERBURY,67601
625-2551
43 M 1902 69 PD
FLANDERS, H ALDEN, 201 EAST 7TH,67601
628-8251
23 M 1902 44 IM
HAIGLER, JAMES P, 234 WEST 11TH,67601
625-2537
13 M 3006 39 FP
HALLING, L WILLIAM, 1300 EAST 13TH,67601
625-5646
27 M S002 57 PATH
HANSA, HANSA, 2503 CANTERBURY,67601
625-2551
45 M 89101 69 OBG
HUTCHISON, GLEN C, 3200 COUNTRY LANE,67601
628-8251
21 M 1902 50 ANES
KANE JR, WILLIAM M, CANTERBURYS CLINIC PA,67601
628-3245
27 M 1001 54 OBG
KIFER, C JAMES, BOX 833,67601
625-6521
45 M 1902 71 DR
LASLEY, MICHAEL B, 2707 VINE SUITE 7,67601
628-3217
45 M 1902 71 GS
MATTICK, IRVIN H, 2818 N VINE,67601
628-8221
18 M 2802 43 ORS
MEDINA, ANIBAL, 2707 VINE,67601
628-1019
34 M 45101 62 U
MYRICK, MICKEY, 2501 CANTERBURY RD,67601
625-2518
42 M 3005 74 FP
NEIL, ROY NEWTON, PO BOX 1019,67601
628-3215
38 M 3005 65 PATH
NEWCOMB, WARD M, 1300 E 13TH,67601
625-5646
47 M 3005 71 PATH
REYNOLDS, JEFFREY C, 2517 CANTERBURY RD,67601
625-7311
39 M 1902 64 ENT
REYNOLDS, LLOYD W, 2110 ASH,67601
-
10 M 3840 34 OO
RICHARDS, DALLAS LEE, 2707 VINE #6,67601
628-3261
49 M 1902 76 IM
SILER, EUGENE T, 1010 DOWNING,67601
628-8218
24 M 1902 52 OO
STUMP, HARL G, 2717 CANAL BLVD,67601
628-2826
39 M 1902 65 GS
WATTS, HARRY E, 1010 DOWNING AVE,67601
628-8218
27 M 702 54 OPH
WEBER, WALLACE N, 2707 VINE SUITE 10,67601
628-3231
43 M 1902 69 O
WENZEL, ANNA MARIE, 204 W 17TH,67601
-
95 F 1902 23 OO
WIEGMAN, HUGH ALAN, BOX 833,67501
628-1808
34 M 1803 60 OPH
WILCOX JR, HOWARD L, PO DRAWER 430,67601
628-8221
44 M 1902 70 ORS

HAYSVILLE—316
(Sedgwick County Society)

KRUPKA, MILES ALBERT, 141 N MAIN,67217
529-2151
49 M 1611 75 FP
MORGAN, NOVA L, 301 W GRAND,67060
225-1371
20 M 3901 50 FP

HERINGTON—913
(Dickinson County Society)

DOZIER, FRED S, 1005 NORTH B STREET,67449
258-2215
10 M 4804 34 FP

HESSTON—316
(Harvey County Society)

DIENER, CLAYTON H, PO BOX 386,67062
327-4122
18 M 1902 54 GS
FRIESEN, FLORENCE V, SHOWALTER VILLA,67062
-
87 F 1611 14 OO
YODER, VERNON E, ROUTE #1,67062
283-2400
31 M 4802 61 P

HIAWATHA—913
(Northeast Kansas Society)

DUCKETT, THOMAS G, 201 MIAMI,66434
742-2161
10 M 1902 34 FP
LARSON, DELBERT L, 314 OREGON,66434
742-2161
30 M 1803 64 FP
MEIDINGER, RAY, 111 S FOURTH,66434
742-2135
03 M 3005 32 FP
SINNING, GARY, 314 OREGON,66434
742-2161
49 M 1902 74 FP

HIGHLAND—913
(Northeast Kansas Society)

CORDER, ROBERT L, ,66035
442-3341
23 M 1902 49 FP

HILL CITY—913
(Northwest Kansas Society)

REDDY, B N, 304 WEST PROUT,67642
674-2255
38 M 49557 66 PD
REDDY, P JAGANNADHA, 80 WALNUT DRIVE,67642
674-2255
42 M 49511 66 GS

HILLSBORO—316
(Marion County Society)

ENS, GERHARD GEORGE, 613 SOUTH MAIN,67063
947-5931
20 M 1902 55 FP
ENS, PETER, 209 SOUTH MAIN,67063
947-3671
14 M 1902 51 FP
FRANZ, ROBERT G, 704 S MAIN,67063
947-3197
33 M 3901 59 FP
JANZEN, HERMAN F, 212 S JEFFERSON,67063
-
91 M 1902 35 GP
LOEWEN, PETER S, SKILLED NURSING UNIT,67063
-
91 M 1902 30 OO

HOISINGTON—316
(Barton County Society)

MOORE, ROBERT, 814 NORTH ELM,67544
653-2151
22 M 3901 53 FP

HOLTON—913
(Jackson County Society)

MOSER, ERNEST C, 438 HILLCREST DR,66436
-
09 M 1902 34 OO
MOSER, M ROSS, 418 WEST FIFTH,66436
364-3116
19 M 1902 47 FP
MOSER, ROY H, 801 IOWA,66436
-
04 M 1902 32 OO
SEELEY, JAMES C, 418 WEST 5TH,66436
364-3116
34 M 1902 64 FP

HORTON—913
(Northeast Kansas Society)

FRANCISCO, EDGAROO, PO BOX 6,66439
486-2646
39 M 74802 57 GP
WOOD, ROBERT D, 105 W 8TH,66439
486-2127
26 M 1902 53 FP

HOWARD—316
(Southeast Kansas Society)

ENRIQUEZ, ROMAN, 114 E WASHINGTON,67349
374-2521
48 M 74801 72 IM

HOXIE—913
(Northwest Kansas Society)

NEUENSCHWANDER, JOHN, 700 MAIN,67740
675-3292
26 M 2802 51 FP
NEUENSCHWANDER, JOHN RAND, 700 MAIN,67740
675-3292
47 M 1902 72 FP

HUGOTON—316
(Seward County Society)

FREDERICK, M F, 1006 S JACKSON,67951
544-2784
20 M 1902 44 FP
LENEVE, ROBERT T, 1006 S JACKSON,67951
544-2784
21 M 3901 46 FP

HUMBOLDT—316
(Southeast Kansas Society)

LONG, EDWARD E, 8TH & NEW YORK,66748
473-2411
21 M 1902 50 FP

HUTCHISON—316
(Reno County Society)

ADAMS JR, MARCUS W, 2101 N WALDRON,67501
663-6121
33 M 3901 59 PD
ALBRIGHT, JEROLD O, 2101 N WALDRON,67501
663-6121
39 M 1902 66 FP
ANTLFINGER, THOMAS J, 2221 E 56TH ST,67501
663-6811
42 M 5606 67 R
BAUER, THOMAS A, 2101 N WALDRON,67501
663-6121
41 M 1902 67 1M
BLANK, JOHN N, WOLCOTT BLDG,67501
663-4271
07 M 1902 38 FP
BORRA, MARIO J, 1 WEST 8TH,67501
662-1751
24 M 2401 47 U
BOS, NORMAN C, 2101 N WALDRON,67501
663-4406
24 M 1611 47 ORS
BRADA, DONALD ROBERT, 308 W 20TH,67501
662-6041
39 M 1902 65 P
BURGER, J DALE, 2101 N WALDRON,67501
663-6121
21 M 1902 46 FP
CASADY, GILBERT N, 39 LINKSLAND DR,67501
663-3657
23 M 1902 53 ANES
CASEY, JAMES, 2101 N WALDRON,67501
663-6121
42 M 3005 69 PD
CHERVEN, PHILIP L, 1100 N MAIN,67501
662-3364
45 M 2501 71 PD
CRAWFORD, ROBERT A, 2101 N WALDRON,67501
663-6121
16 M 1601 42 FP
DEPENBUSCH, FRANCIS L, 1708 E 23RD,67501
663-7187
38 M 1902 65 OPH
ECKART, DE MERLE E, 312 WOLCOTT BLDG,67501
662-4921
14 M 1902 40 P
FALTER, RICHARD T, 1708 E 23RD ST,67501
663-7187
38 M 1902 67 OPH

FERNIE, ROBERT W, 1100 N MAIN,67501
663-1136
06 M 702 34 1M
FOSS, DANIEL C, 2101 N WALDRON,67501
663-6121
43 M 1902 69 GE
GOODPASTURE, WILLARD C, 2101 N WALDRON,67501
663-6121
10 M 1602 36 OO
GRAVES, KATHRYN, 2101 N WALDRON,67501
663-6121
49 F D
HEDRICK, KENNETH E, 2101 N WALDRON,67501
663-6121
27 M 1902 53 GS
HENNEBERRY, MICHAEL, 2101 N WALDRON,67501
663-6121
M 1606 U
HOLDERMAN, WALLACE D, 2101 N WALDRON,67501
663-4406
28 M 1902 54 ORS
JARROTT, JOHN B, 1100 N MAIN,67501
669-0191
16 M 1902 40 ORS
KLOSTERHOFF, BRUCE E, 1701 E 23RD,67501
669-0619
M P
LETTNER, HANS T, 720 N MAIN,67501
662-7801
23 M 40716 49 PATH
LUKENS, DAVID, 2101 N WALDRON,67501
663-1136
24 M 2307 48 1M
MCCOY, CHARLES T, 310 WOLCOTT BLDG,67501
662-0121
16 M 1902 41 OPH
MCMULLEN, JOSEPH E, 2101 N WALDRON,67501
663-6121
33 M 1902 62 GS
MULL, JOHN C, 2101 N WALDRON,67501
663-6121
34 M 1902 61 ORG
NEEL, WILBUR B, 2101 N WALDRON,67501
663-6121
24 M 1902 59 1M
NEUSCHAFER, DARREL R, 2101 N WALDRON,67501
663-6121
48 M 1902 74 OBG
NUNEMAKER, MARION E, 2415 N MAIN,67501
662-5391
21 M 1902 46 ANES
OPENSHAW, CALVIN R, 1100 N MAIN,67501
662-3305
21 M 4901 44 GS
PAINE, GEORGE E, 220 W 23RD,67501
94 M 1606 19 OO
PEASE, GARY L, 521 WILEY BLDG,67501
662-4458
41 M 3005 67 OTO
PERKINS, JACK L, 2101 N WALDRON,67501
663-6121
24 M 1902 53 FP
SCHROLL, JACK C, 2101 N WALDRON,67501
663-6121
24 M 1902 49 OBG
SHAW JR, JAMES W, 720 N MAIN,67501
662-7801
40 M 1902 65 PATH
SHEARS, ROBERT N, 1100 N MAIN,67501
662-3364
20 M 1902 44 PD
STAFFORD, ROBERT W, 2101 N WALDRON,67501
663-6121
43 M 2101 69 1M
STENSAAS, CARL O, 401 WOLCOTT BLDG,67501
663-9147
10 M 1902 38 D
STOUT, JAMES M, 2101 N WALDRON,67501
663-6121
29 M 1902 55 FP
SUMNER, JOYCE R, 2027 N MADISON,67501
663-0822
26 F 1902 51 ANES
SUMNER, MARION M, 2027 N MADISON,67501
663-6121
26 M 1902 52 1M
TWEITO, DAVID H, 1100 N MAIN,67501
662-3364
38 M 1803 64 PD
VON RUDEN, WILLIAM J, 2100 N WALDRON,67501
662-3306
26 M 1511 52 GS

WILLIAMSON, JOHN, 1100 N MAIN, 67501
669-0191
35 M 2501 60 ORS
WORTMAN, JACK A, 2101 N WALDRON,67501
663-6121
34 M 1902 62 IM

INDEPENDENCE—316
(Southeast Kansas Society)

BAIR, ALBERT E, PO BOX 925,67301
331-7650
16 M 1902 44 GS
BARBERA, PORTER E, 800 WEST CHESTNUT,67301
331-4400
19 M 4707 45 FP
BEAHM, EDGAR H, 307 PROFESSIONAL BLDG,67301
331-2311
13 M 1902 44 FP
BULLOCK, HAROLD D, PO BOX 746,67301
331-5550
06 M 1902 33 IM
CHAPPUIE, WILLIAM G, 800 CHESTNUT WEST,67301
331-5440
24 M 1902 51 FP
EMMOT, WILLIAM W, 800 WEST CHESTNUT,67301
331-6350
45 M 1902 71 IM
EMPSON, CHARLES L, 400 N 14TH,67301
331-6019
37 M 1902 68 FP
GAREY, WILLIAM JOHN, MERCY HOSPITAL,67301
331-2200
44 M 6501 DR
KNUTH, KENNETH L, MERCY HOSPITAL,67301
331-2200
22 M 1902 50 R
MYERS JR, EARL B, BOX 548,67301
331-3420
32 M 2803 64 GS
OSBORN DO, ROBERT M, 800 W CHESTNUT,67301
331-2070
M GP
ROBINSON, EDGAR L, 209 NORTH 6TH,67301
331-1450
15 M 1902 42 FP
SHEIKH, MASOOD A, 900 W MYRTLE,67301
331-2803
44 M 70402 66 GS
STRAHM, WAYMER J, RT 1,67301
331-1748
29 M 1902 62 P
SUTTON, ROBERT E, PO BOX 1003,67301
331-7700
46 M 1902 72 FP

IOLA—316
(Allen County Society)

COPENING, TELL B, 305 N WASHINGTON,66749
365-2134
43 M 1902 69 FP
DETAR, GEORGE F, 219 W MADISON,66749
365-3712
28 M 1902 57 GS
DICK, WILLIS G, BOX 826,66749
365-2131
13 M 512 41 GS
HANSON, DAVID C, 826 E MADISON,66749
365-2131
46 M 512 73 FP
LENSKI JR, FRANCIS X, 206 S JEFFERSON,66749
365-3901
26 M 1606 49 FP
MYERS, W EUGENE, 211 S STREET,66749
365-3732
12 M 1902 46 FP
PEES, GERALD B, 219 W MADISON,66749
365-5175
15 M 1902 43 GS
SCHMAUS, LYLE F, 202 E STREET,66749
99 M 1803 26 OO

JETMORE—316
(Ford County Society)

O'SHEA, JAMES G, BOX 545,67854
357-8321
18 M 3901 48 FP

JEWELL—913
(Mitchell County Society)

PLOWMAN, CARL W, ,66949
428-3511
99 M 1606 26 OO

JUNCTION CITY—913
(Geary County Society)

BOLLMAN, CHARLES S, PO BOX 397,66441
762-4575
41 M 3901 66 GS
BRETHOUR, LESLIE J, PO BOX 49,66441
238-4151
13 M 3005 39 FP
BUNKER JR, HERBERT L, 1106 ST MARYS RD,66441
238-5131
20 M 1606 45 FP
COPELAND, GARY A, GEARY COMMUNITY HOSP,66441
762-2387
42 M 1902 68 R
LABHSETWAR, S A, MEDICAL ARTS BLDG,66441
762-4147
39 F 49528 64 OBG
MACE, RONALD D, PO BOX 163,66441
762-4884
42 M 3901 74 FP
MINNICK, CHARLES V, 602 NORTH JEFFERSON,66441
762-2051
08 M 2105 35 FP
O'DONNELL, HARRY E, 1106 ST MARYS RD,66441
762-4947
14 M 4113 42 FP
SCOTT, ALEX, 507 WEST 6TH,66441
238-2518
23 M 5605 48 FP
WRIGHT, STANLEY E, 1106 ST MARYS RD,66441
762-4884
47 M 3901 74 FP

KANSAS CITY, KANSAS—913
(Wyandotte County Society)

ABDOU, NABIH I, KU MEDICAL CENTER,66103
588-6586
34 M 33002 58 A
ALEXANDER, CHARLES E, 600 NEBRASKA,66101
321-6670
43 M 401 70 OBG
ALEXANDER, CLYDE W, 1514 NORTH 5TH,66101
281-4380
96 M 4707 23 FP
ALGIE, WILLIAM H, 907 N 7TH,66101
371-5079
02 M 1902 27 IM
ALLEN, MAX S, K U MED CENTER,66103
588-6988
11 M 1902 37 IM
ALLEN, WILLIAM R, 9201 PARALLEL PARKWAY,66112
334-4110
22 M 1902 46 R
AMARF, MAMMO, KUMC,66103
588-6031
36 M 60501 61 IM
ARAKAWA, KASUMI, KU MEDICAL CENTER,66103
588-6670
26 M 57211 53 ANES
ARENAL, ANGELA C, BETHANY HOSPITAL,66102
281-8400
38 F 35204 60 ANES
ASHER, MARC A, K U MED CENTER,66103
588-6130
36 M 1902 62 ORS
ATKINS JR, FLOYD L, K U MED CENTER,66103
588-6015
43 M 5104 69 CD
BAILIE, MICHAEL DAVID, KU MED CENTER,66103
355-6339
36 M 1720 64 PNP
BARNHORST, DONALD A, KUMC,66103
588-6107
37 M 2834 63 CDS
BASS JR, LEWIS N, 1975 N 5TH,66101
321-2320
21 M 1902 45 PD
BATNITZKY, SOLOMON, K U MED CENTER,66103
588-6835
40 M 83601 64 OR
BECKER, LESLIE E, 600 NEBRASKA,66101
342-4010
23 M 1003 46 U
BELT, ROBERT JULIAN, K U MED CENTER,66103
588-6023
45 M 702 71 MO

BERGIN, JAMES J, BETHANY MED CENTER,66102
281-8767
28 M 2407 54 IM

BICHLMEIER, FRANKLIN G, 155 S 18TH,66102
371-6800
33 M 1902 58 CDS

BIGONGIARI, LAWRENCE R, KU MED CENTER,66103
588-6800
44 M 1611 69 OR

BILLINGSLEY, THAD H, 155 S 18TH,66102
321-3844
41 M 1902 66 P

BOGGAN, MICHAEL D, 155 S 18TH,66102
371-6800
40 M 1201 67 CDS

BOLINGER, ROBERT E, KU MEDICAL CENTER,66103
588-6022
19 M 1902 43 END

BOLTON, VICTOR E, 155 SOUTH 18TH,66102
371-2020
24 M 1902 48 DBG

BOSILEVAC, FRED N, 155 S 18TH,66102
342-4843
16 M 1902 44 OPH

BRACKETT JR, CHARLES E, KU MED CENTER,66103
588-6117
20 M 3501 44 NS

BRILLHART, MAXINE T, 1610 WASHINGTON BOULEVARD,66102
321-4800
15 F 1902 50 FP

BROOKS, WILLIAM HENRY, 155 S 18TH,66102
371-4343
49 M 1902 74 R

BUBB, STEPHEN K, 155 S 18TH,66102
371-6802
48 M 1902 74 ORS

BURGER, WILLIAM E, 355 NEW BROTHERHOOD BLDG,66101
371-1017
21 M 3006 51 GS

CALDERON, JAIME, 4631 ORVILLE,66102
596-1185
39 M 26401 66 IM

CAMERON, WILLIAM J, KU MEDICAL CENTER,66103
588-6200
29 M 2501 54 OBG

CARLIN, JAMES WILLARD, 39 E RAINBOW,64131
588-6670
51 M 1902 76 ANES

CARPENTER, PAUL R, 155 SOUTH 18TH,66102
371-6800
24 M 1902 50 TS

CASTEEL, CHARLES K, 155 SOUTH 18TH,66102
342-7233
34 M 3901 59 U

CHALIAN, ALEXANDER R, 2648 MINNESOTA AVENUE,66102
-
03 M 3509 37 DD

CHANG, CHAE H, KU MED CENTER,66103
588-6807
29 M 58301 53 R

CHIN, TOM D, KUMC - HUMAN ECOLOGY DEPT,66103
588-7175
22 M 2501 43 ID

CHO, CHENG T, K U MED CENTER,66103
588-6302
37 M 38501 62 PO

CHONKO, ARNOLD M, KU MED CENTER,66103
588-6969
43 M 3840 69 NEP

CHUN, CHUNG S, K U MED CENTER,66103
588-6324
30 F 58301 53 PD

COALE, LLOYD H, 5020 GREELEY,66104
-
13 M 1902 43 DD

COOK, JAMES D, KU MED CENTER,66103
588-6031
36 M 6505 60 HEM

COOKE, ALLAN R, KUMC DEPT OF MED,66103
588-6990
36 M 14303 58 GE

COPELAND, SHARON LAVARNE, KU MED CENTER,66103
588-7535
50 F 1902 75 FP

COX III, IRA L, 155 SOUTH 18TH,66102
371-4343
43 M 1902 68 OR

COX JR, WALLACE F, 4631 ORVILLE,66102
287-3600
30 M 1902 56 GS

CROCKETT, CHARLES A, 155 S 18TH,66102
342-2200
19 M 401 44 OPH

CULP, LOUIS M, 1645 WASHINGTON BLVD,66102
371-1077
24 M 1902 53 FP

CURTIS, MIRJANA R, KU MED CENTER - OB/GYN,66103
588-6237
46 F 95702 69 OBG

DARR, RICHARD B, 147 APACHE TRAIL W,66106
676-2214
42 M 3401 70 IM

DAVIS, CHRISTOPHER G, 219 HURON BLDG,66101
321-9313
09 M 1902 39 FP

DAY, HUGHES W, BETHANY MED CENTER,66102
281-8856
15 M 1902 39 IM

DE SMET, ARTHUR AUGUST, KU MED CENTER - DIAG RAD,66103
588-6800
47 M 2501 72 OR

DEITZ, MICHAEL R, 155 S 18TH,66102
342-2222
32 M 4101 58 OPH

DEMOTT, WAYNE R, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
34 M 4002 59 PATH

DIALLO, GASTON I, 600 NEBRASKA,66117
281-2888
35 M 86905 64 GE

DICK, ARTHUR R, KUMC,66103
588-6985
34 M 2301 65 N

DIEDERICH, DENNIS A, K U MEDICAL CENTER,66103
588-6981
36 M 2834 61 IM

DONNELLY, FRANCIS M, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
48 M 1902 74 ANES

DUJOVNE, CARLOS A, K U MED CENTER,66103
588-6026
37 M 13201 61 CP

DUNN, MARVIN I, KU MED CENTER,66103
588-6015
27 M 1902 54 CD

DVORAK, EDUARD, KU MED CENTER - RADIOTHER,66103
588-7350
43 M 28601 66 R

EGEA, FERNANDO M, 8919 PARALLEL PARKWAY,66112
334-3400
37 M 13206 62 N

FABIAN, CAROL J, K U MED CENTER,66103
588-6029
46 F 1902 DN

FITZPATRICK, M ROBERT, 1610 WASHINGTON BLVD,66102
321-4800
20 M 1803 44 FP

FIXLEY, MARK S, K U MED CENTER,66103
588-6044
45 M 1902 72 PUD

FLOERSCH, HUBERT M, 155 S 18TH,66102
371-2020
08 M 1902 35 OBG

FORET, JOHN D, KU MED CENTER,66103
588-6147
26 M 1602 53 U

FOX, DEANNA K, K U MED CENTER,66103
588-6670
48 F 1902 74 ANES

FOX, HOWARD A, K U MED CENTER,66103
588-6337
33 M 3501 62 PD

FRANCISCO, W DAVID, 155 S 18TH,66102
371-6802
21 M 1902 44 ORS

FRIESEN, STANLEY R, KU MED CENTER,66103
588-6108
18 M 1902 43 GS

GERJARUSAK, PRAPAS, 8919 PARALLEL SUITE 208,66112
788-9604
46 M 89101 71 IN

GIAP, HAI PHUC, KU MED CENTER DEPT OF PSY, 66103
588-6400
44 M 94101 73 P

GILHOUSEN, FREDERIC M, 1029 N 32ND,66102
281-5252
40 M 1902 66 ORS

GILMORE, CLARENCE A, 600 NEBRASKA,66101
321-6670
31 M 4707 60 OBG
GODFREY, WILLIAM A, KUMC,66103
588-6600
38 M 1902 65 OPH
GOERTZ, KENNETH K, 222 HC MILLER KUMC,66103
588-6311
50 M 1902 75 PO
GOERTZ, LEO R, 155 S 18TH ST,66102
371-4343
22 M 1902 52 R
GOODWIN, DONALD W, KUMC,66103
588-6402
31 M 1902 64 P
GOTO, HIROSHI, KUMC - ANES DEPT,66103
588-6670
42 M 57241 67 ANES
GRADY, KENNETH L, 155 S 18TH,66102
321-3844
36 M 1902 69 P
GRANTHAM, JARED J, K U MED CENTER,66103
588-6983
36 M 1902 62 NEP
GREENBERGER, N J, KUMC,66103
588-6001
33 M 3806 59 IM
GRIPKEY, CLARENCE A, 707 NORTH 7STH,66112
-
05 M 1902 30 OD
GRUENDEL, RICHARD A, 1029 N 32ND,66102
281-5252
29 M 1902 55 ORS
GRUENDEL, VIRGINIA T, 6926 GARFIELD,66102
299-2787
30 F 1902 55 FP
HAAS, CHARLES D, K U MED CENTER,66103
588-6029
39 M 1902 68 IM
HALL III, THOMAS BRYAN, KU MED CENTER,66103
588-6400
43 M 2802 69 P
HANCOCK, ALAN C, 9201 PARALLEL,66112
299-1474
35 M 1902 65 FP
HARA, GLENN S, KU MED CENTER,66103
588-6200
43 M 502 69 OBG
HARDIN, CREIGHTON A, KU MED CENTER,66103
588-6106
18 M 5605 43 GS
HARTMAN, CHARLES R, K U MED CENTER,66103
588-6111
37 M 1902 66 IM
HARTMAN, GERALD V, KUMC,66103
588-6815
20 M 1902 45 TR
HENNING JR, HAROLD JOHN, 4116 BOOTH,66103
-
55 M 1902
HERMRECK, ARLO S, K U MED CENTER,66103
588-7232
38 M 1902 65 GS
HINTHORN, DANIEL R, KUMC,66103
588-6035
41 M 1902 67 IM
HITCHCOCK, C THOMAS, 155 SO 18TH SUITE 200,66102
371-2900
M 1902 78 GS
HODGES, GLENN R, K U MED CENTER,66103
588-6035
41 M 1602 67 ID
HOLOCRAFT, JACQUELYNE, 255 NEW BROTHERHOOD BLDG,66101
321-1161
36 F 2105 63 ENT
HOLLEMAN JR DO, JAMES F, 667 S 55TH ST,66106
287-1414
38 M 2878 68 FP
HOLMES, FREDERICK F, KUMC,66103
588-6987
32 M 5404 57 IM
HOLMES, GRACE E, KUMC,66103
588-6325
32 F 5404 57 PD
HOOGSTRATEN, BARTH, K U MED CENTER,66103
588-6029
24 M 66001 55 ON
HORTON, WILLIAM A, K U MEDICAL CENTER,66103
588-6043
45 M 1902 71 IM
HUERTER, QUENTIN C, WYANDOTTE MED BLDG STE226,66112
299-8800
31 M 1902 59 OPH
HURWITZ, ARYEH, KU MED CENTER,66103
588-6026
36 M 2802 61 IM
IBARRA, RICHARD C, 754 PACIFIC,66101
342-3969
26 M 64902 57 FP
INGRAM, JOHN E, 1428 S 32ND,66106
384-1630
24 M 3006 56 FP
INNES, ROBERT C, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
25 M 2802 49 R
JACOBS, DAVID S, 8929 PARALLEL PARKWAY,66112
334-2500
31 M 2501 56 PATH
JACOBS, RAE R, K U MED CENTER,66103
588-6504
36 M 3505 62 ORS
JAHANIAN, DARYOUSH, 155 S 18TH,66102
371-2020
40 M 51701 64 OBG
JAYARAM, MARANDAPALLI R, 8919 PARALLEL,66112
492-6200
42 M 49509 65 PD
JEFFRIES, RHONDA DEBERT, 436 STATE AVE,66101
321-3055
51 F 1902 75 PD
JEWELL, WILLIAM R, KU MED CENTER,66103
588-6112
35 M 1611 61 GS
JOHNSON, JOHN E, BETHANY MED CTR,66102
281-8815
17 M 4705 43 PATH
JONES JR, HERMAN H, 600 NEBRASKA,66101
342-4010
25 M 4707 54 GS
KALIVAS, JAMES T, KU MED CENTER,66103
588-6028
38 M 502 63 O
KENNEDY, JAMES A, K U MED CENTER,66103
588-6000
35 M 2834 61 IM
KEPES, JOHN J, K U MED CENTER - PATH DEP,66103
588-7076
28 M 47301 52 PATH
KERBY, GERALD R, KU MED CENTER,66103
588-6044
32 M 1902 58 PUD
KESTENBAUM, THELDA M, K U MED CENTER,66103
588-6028
48 F 5101 73 O
KIM, JONG M, KUMC,66103
588-6670
40 M 58302 64 ANES
KING, CHARLES R, K U MEDICAL CENTER,66103
588-6200
47 M 1902 71 OBG
KIRCHNER, NANCY, KU MED CENTER,66103
588-6800
44 F 4802 73 POR
KIRCHNER, FERNANDO R, 155 S 18TH SUITE 270,66102
371-7333
30 M 64901 55 OTO
KRANTZ, KERMIT E, KU MED CENTER,66103
588-6201
23 M 1606 48 OBG
KWEE, SIDE T, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
36 F 1720 63 PATH
KYNER, JOSEPH L, KU MED CENTER,66103
588-5554
34 M 1902 60 END
LAING, ROBERT R, 155 S 18TH ST,66102
371-4301
37 M 1643 61 GE
LANSKY, LESTER L, K U MED CENTER,66103
588-6300
33 M 2604 65 CHN
LANSKY, SHIRLEY B, K U MED CENTER,66103
588-6400
35 F 2504 60 CHP
LAYBOURNE JR, PAUL C, KU MED CENTER,66103
588-6475
19 M 3509 44 CHP
LEAKE III, HUNTER C, K U MED CENTER - PED DEPT,66103
956-1100
37 M 2105 63 PD
LEE, JAE M, 4631 ORVILLE - SUITE 109,66102
677-3555
40 M 58302 65 GS
LEE, KYO R, KUMC,66103
588-6800
33 M 58302 59 R

LEE JR, JAMES G, 155 S 18TH,66102
371-2330
18 M 1902 44 OBG
LEMOINE JR, ALBERT N, K U MED CENTER,66103
588-6600
18 M 2802 43 OPH
LEO, WILLIAM A, K U MED CENTER,66103
588-6109
22 M 1902 48 GS
LEVINE, ERROL, K U MED CENTER,66103
588-6800
41 M 83601 64 DR
LIEBERMAN, BRUCE IRWIN, KU MED CENTER,66103
588-6300
49 M 3819 74 PD
LINDSLEY, CAROL B, K U MED CENTER,66103
588-5907
41 F 5404 68 PD
LINDSLEY, HERBERT B, K U MED CENTER,66103
588-6008
40 M 1902 66 RHU
LINSHAW, MICHAEL A, K U MED CENTER,66103
588-6300
40 M 4109 66 PD
LISKOW, BARRY IRWIN, KU MED CENTER DEPT OF PSY,66103
588-6412
43 M 3501 68 P
LIU, CHIEN, K U MED CENTER,66103
588-6035
21 M 24217 47 ID
LLOYD, HARVEY L, 3200 STRONG AVE,66106
831-1111
08 M 1803 36 FP
LOTITO, CARLOS A, 4631 ORVILLE,66102
596-1185
29 M 13201 56 IM
LOWMAN, JAMES T, KUMC,66103
588-5701
31 M 401 58 PD
LUKERT, BARBARA P, KU MED CENTER,66103
588-5554
34 F 1902 60 END
LYNCH, SEAN R, KU MED CENTER,66103
588-6031
38 M 83601 61 HEM
MACARTHUR, RICHARD IAN, KUMC 39TH AT RAINBOW,66103
588-6100
46 M 1902 73 GS
MAKLAD, M NABIL, K U MED CENTER - RAD DEPT,66103
588-6886
38 M 33003 61 R
MANGOLD, JOEL JOYCE, KU MED CENTER,66103
588-6670
50 M 1902 76 ANES
MANI, MANI M, KUMC,66103
588-6136
37 M 49527 60 PS
MANSFIELD, CARL M, K U MED CENTER - RM 1178,66103
588-7350
28 M 1003 56 TR
MARTIN, NORMAN L, K U MED CENTER,66103
588-6800
36 M 1902 62 DR
MARVIN, NORMAN G, KUMC 39TH AT RAINBOW,66103
588-6394
29 M 1902 56 FP
MASTERS, FRANCIS W, KU MED CENTER,66103
588-6142
20 M 3545 45 PS
MASTERSON, BYRON J, KUMC,66103
588-6216
33 M 2802 58 GYN
MATHEWSON, HUGH S, KUMC,66103
588-6675
21 M 1902 44 ANES
MATTIOLI, LEONE, KUMC,66103
588-6311
32 M 56115 56 PDC
MCCARTHY, ROBERT P, 155 S 18TH,66102
342-7233
25 M 2834 53 U
MEBUST, WINSTON K, KU MED CENTER,66103
588-6148
33 M 5404 58 U
MEDEARIS SR, DONALD N, 3100 MINN AVE,66102
-
01 M 2401 27 OD
MEEK JR, JOSEPH C, KU MED CENTER,66103
588-6023
31 M 1902 57 IM
MELHORN, J MARK, 3428 E CENTRAL,67208
-
53 M 1902 80

MESINA, ROLANDO R, 1102 MINNESOTA AVE,66102
371-3829
37 M 74801 61 GS
MILITANO, THOMAS CHARLES, KU MED CENTER,66103
588-6123
42 M 3806 63 TS
MINER JR, PHILIP B, K U MED CENTER,66103
588-6990
46 M 702 71 GE
MOELLER, DONALD D, 155 S 18TH,66102
371-4301
34 M 1902 60 GE
MOFFAT, ROBERT E, K U MED CENTER,66103
588-6800
42 M 1902 68 DR
MOORE, WAYNE V, K U MED CENTER,66103
588-6336
42 M 2604 70 PD
MORFFI, RAUL R, 1225 N 78TH,66112
287-8300
25 M 27501 51 IM
MORRISON, RICHARD A, KU MED CENTER,66103
588-6815
34 M 1902 60 TR
MULLEN JR, CLIFFORD J, 9201 PARALLEL,66112
334-4250
38 M 1902 64 OPH
MULLEN SR, CLIFFORD J, 1828 WASH BLVD,66102
-
98 M 3006 23 OD
NEFF, JAMES R, KU MED CENTER,66103
588-6198
40 M 1902 66 ORS
NEIGHBOR, ERNEST G, 1420 S 42ND,66106
831-1100
06 M 1902 33 FP
NEIGHBOR, ERNEST H, 1420 SOUTH 42ND ST,66106
831-1100
40 M 1902 66 ORS
NEIGHBOR, GAYLORD P, 1420 SOUTH 42ND,66106
831-1100
13 M 1902 41 FP
NELSON, WILLIAM PAUL, KU MED CENTER,66103
588-6015
30 M 1002 56 CD
NEWKIRK, DARREL D, 619 ANN AVE,66101
321-4803
42 M 2101 67 PH
NIEBELINK, LARRY WAYNE, 155 S 18TH,66102
371-2330
48 M 2803 75 OBG
NOHE, PHILIP C, 266 NEW BROTHERHOOD BLDG,66101
321-4422
17 M 1902 42 ORS
NORRIS, CHARLEY W, KUMC,66103
588-6700
33 M 1902 64 OTO
NOTHNAGEL, ARNOLD F, 501 WESTVALE,66102
-
15 M 1902 39 OD
NUNEZ, JULIAN, 4631 ORVILLE #209,66102
299-2088
30 M 27501 60 FP
O'BOYNICK II, PAUL LEONARD, KU MED CENTER,66103
588-5000
48 M 1902 73 NS
OLDFIELD, RAY W, 4631 ORVILLE,66102
287-3537
45 M 1902 72 FP
OTHMER, EKKEHARD, K U MEDICAL CENTER,66103
588-6440
33 M 40721 66 P
OXLER JR, JOHN EDWARD, 155 S 18TH,66102
321-2974
46 M 1902 72 IM
PALMER, MARVIN M, 4137 N 110TH,66109
727-6000
45 M 702 71 OBG
PARDO, LILLIAN G, KU MED CENTER,66103
588-5909
39 F 74807 62 PDN
PARDO, MANUEL P, K U MEDICAL CENTER,66103
588-6464
35 M 74801 62 P
PAREKH, AJITKUMAR M, 6013 LEAVENWORTH RD,66104
299-2069
47 M 49501 71 PUD
PARK, CHAN H, K U MEDICAL CENTER,66103
588-6029
36 M 58302 62 IM

PARRA, MIGUEL D, 6013 LEAVENWORTH RD,66104
299-2088
37 M 84710 64 FP
PASCHINO, ANTONIO R, 4631 ORVILLE #215,66102
287-7100
22 M 13201 60 P
PATAK, RAMACHANDRA V, KU MEDICAL CENTER,66103
588-6983
43 M 49509 66 N
PAZELL, JOHN A, 4631 ORVILLE,66102
287-6464
40 M 2501 66 DRS
PECANA, MANUEL C, BETHANY MED CENTER,66102
281-8896
45 M 74801 69 ANES
PERKINS, WILLIAM GENE, C & Y CLINIC,66101
321-3055
49 M 1902 74 PD
PERRY JR, LAWRENCE L, KUMC 39TH AT RAINBOW,66103
588-6522
34 M 1902 59 FP
PETELIN, JOSEPH B, 3227 N 54TH,66104
588-6100
49 M 1902 76 GS
PETERS, GLENN R, 155 SOUTH 18TH,66102
371-6800
12 M 1902 37 GS
PIERCE, GEORGE E, KUMC - PO BOX 255,66103
588-6115
33 M 2307 60 TS
PISCHKE, FRANK J, 155 SOUTH 18TH,66102
321-1161
36 M 1902 62 OTO
PDL, P ALBERT, 6013 LEAVENWORTH RD,66104
287-7100
36 M 84710 67 P
PORTER, DAVID M, 4517 TROUP,66102
287-8800
39 M 4707 64 PD
POTTER, ROBERT L, 1969 N 33RD,66102
321-0341
38 M 1902 64 IM
POWERS, G ROBERT, 8919 PARALLEL PKWY,66112
299-1474
33 M 1902 65 FP
PREMSINGH, NALINI G, 4631 ORVILLE #202,66102
596-2000
39 F 49508 65 CO
PRESTON, DAVID F, KU MED CENTER,66103
588-6810
33 M 3841 59 NM
PRETZ, JAMES B, 1610 WASHINGTON BOULEVARD,66102
342-2442
24 M 1902 47 FP
PRICE, JAMES GORDON, K U MED CENTER,66103
588-6510
26 M 702 47 FP
PROUD, G ONEIL, KU MEDICAL CENTER,66103
588-6700
13 M 2802 39 OTO
PUGH, DAVID M, K U MED CENTER,66103
588-6015
29 M 801 58 CO
QUAISON, EMMELINE P, KUMC,66103
588-6463
45 F 74802 69 P
QUINN, CHARLES E, 1 GATEWAY CTR 4TH & STATE,66101
321-3355
43 M 4707 68 OBG
RECKLING, FREDERICK W, KUMC,66103
588-6129
34 M 3545 59 ORS
REDDY, EASHWER K, K U MED CENTER - RAD DEPT,66103
588-7350
44 M 49597 68 TR
REDFORD, JOHN W B, K U MED CENTER,66103
588-6777
28 M 6501 53 PM
REES, RONALD JOSEPH, 155 S 18TH,66102
371-4343
46 M 3006 72 OR
REGIER, HENRY L, 2000 WASHINGTON BOULEVARD,66102
-
81 M 1902 07 OD
RHODES, JAMES B, KU MEDICAL CENTER,66103
588-6019
28 M 1902 58 GE
RHODES, MARTIN L, 1969 N 33RD,66102
321-0341
47 M 1902 72 IM
RICE JR, FREDERICK A, 1029 N 32ND ST,66102
281-5252
36 M 4812 63 ORS

RICK JR, GREGORY GP, 155 S 18TH ST,66102
371-4301
40 M 1902 66 GE
RILEY, RAY B, 2020 ORVILLE,66102
-
06 M 1902 36 OD
RISING, JESSE D, KU MEDICAL CENTER,66103
588-4488
14 M 1902 38 IM
ROBINSON, DAVID W, KU MEDICAL CENTER,66103
588-6136
14 M 4101 38 PS
ROBINSON, JOHN O, K U MED CENTER,66103
588-6670
48 M 1902 74 ANES
ROBINSON, RALPH G, KU MEDICAL CENTER,66103
588-6810
37 M 1902 62 NM
ROBISON, JAMES T, KUMC,66103
588-6600
21 M 4812 45 OPH
RODRIGUEZ, RAUL G, 6013 LEAVENWORTH RD,66104
299-2525
42 M 26401 68 P
RODK, LEE E, 4116 STRONG,66106
831-2834
09 M 1902 38 FP
ROSENTHAL, STANTON J, K U MED CENTER,66103
588-6800
46 M 1902 71 OR
ROTH, ALAN E, BETHANY HOSPITAL 51 N 12,66102
281-8814
35 M 1902 62 PATH
RUBIN JR, BEN, 132 S SEVENTEENTH,66102
371-2561
37 M 3005 61 PD
RUTH, WILLIAM E, K U MED CENTER,66103
588-6044
26 M 1902 53 PUD
RYAN, MICHAEL J, 764 NEW BROTHERHOOD BLDG,66101
342-7070
14 M 2834 37 OTO
SAVIN, VIRGINIA J, KU MED CENTER,66103
588-6983
44 F 4112 70 IM
SCHIMKE, R NEIL, KU MED CENTER,66103
588-6043
35 M 1902 62 IM
SCHOTLAND, EDWARD S, 1300 N 78TH STE 303,66112
334-5250
36 M 86901 66 O
SCHREPFER, ROSEMARY, KU MED CENTER,66103
588-6200
22 F 1902 47 OBG
SCHWEGLER, RAYMOND A, 8919 PARALLEL PKWY,66112
492-6200
37 M 1902 63 CO
SCHWORM, CURTIS P, 155 SOUTH 18TH,66102
371-4343
47 M 3005 73 OR
SERERES, EDGAR P, 662 NEW BROTHERHOOD BLDG,66101
321-2501
15 M 1902 39 FP
SHERMAN, ROBERT P, 9201 PARALLEL AVE,66112
334-0040
34 M 1902 63 PUD
SHUSS, JOHN LOGAN, 3816 STATELINE,66103
588-5000
49 M 1902 75 GS
SIFERS, EARL C, 155 S 18TH,66102
371-2900
24 M 1902 47 GS
SIFERS, TIMOTHY M, 155 S 18TH,66102
371-2900
48 M 1902 74 GS
SILVERS, ALVIN, 1702 SW BOULEVARD,66103
236-9691
18 M 1902 45 FP
SKIKNE, BARRY S, KU MED CENTER,66103
588-6031
45 M 83601 61 HEM
SMITH, STEPHEN O, K U MED CENTER,66103
588-6300
45 M 3006 71 PD
SOUCEK, CHARLES O, 155 S 18TH,66102
371-4343
31 M 3005 56 R
SPAULDING, JOHN S, KU MED CENTER,66103
588-5900
29 M 1602 59 PD

SPEER, LELAND, 1022 HOEL PARKWAY,66102
321-1040
12 M 1902 36 PD
STECHSCHULTE, DANIEL J, K U MED CENTER,66103
588-6008
36 M 2834 62 A
STEELE, CLARENCE H, 255 BROTHERHOOD BLDG,66101
321-1161
14 M 1902 40 OTO
STEINZEIG, SHERMAN M, 155 S 18TH,66102
621-1151
25 M 1902 52 CO
STEPHENS, RONALD L, KUMC,66103
588-6029
39 M 1902 65 IM
STEVENSON, CHARLES E, KUMC,66103
588-6670
19 M 1902 44 ANES
STEWART, DAVID R, K U MED CENTER,66103
588-6124
38 M 3545 64 POS
STUBBLEFIELD, CHARLES T, 155 S 18TH,66102
371-2330
32 M 1902 58 OBG
SULLIVAN, TOMMY GRAY, KU MED CENTER-3C- OB/GYN,66103
588-6208
44 M 1902 71 OBG
TAYLOR, THOMAS L, 155 S 18TH ST,66102
371-2900
40 M 1902 66 GS
TEMPLETON, ARCH W, K U MEDICAL CENTER,66103
588-6805
32 M 3005 57 R
THEROU, LEONA F, K U MEDICAL CENTER,66103
588-6302
41 F 6701 67 PD
THOMAS, JAMES H, K U MED CENTER,66103
588-5901
41 M 2012 66 GS
THOMAS, THOMAS V, 211 INDIAN SPRINGS MED BL,66102
287-2600
37 M 49549 61 COS
THOMPSON, DANNIE M, ONE GATEWAY CENTER,66101
321-3355
35 M 4707 64 OBG
TOALSON, WILLIAM B, 155 S 18TH ST,66102
371-3515
37 M 1902 63 CO
TRUEWORTHY, ROBERT C, KU MED CENTER,66103
588-5701
40 M 2802 66 PD
UTLEY, JAMES HARMON, 51 N 12TH,66102
281-8880
51 M 1605 74 EM
VAN THULLENAR, PHILIP A, BETHANY MED CENTER,66102
281-8834
31 M 2834 57 PATH
VARGHESE, GEORGE, K U MEDICAL CENTER,66103
588-6798
44 M 49509 69 PM
VATS, TRIBHAWAN S, KUMC,66103
588-5701
40 M 49529 63 PD
VILLANUEVA, CESAR L, K U MED CENTER,66103
588-6200
39 M 74802 65 OBG
VILLARANTE, FE TEJERO, KU MED CENTER,66103
588-6795
28 F 74807 63 RM
VISSER, VALYA E, KU MED CENTER,66103
588-6337
47 F 1803 73 PD
WADDELL, BILL D, 155 SOUTH 18TH,66102
321-0386
31 M 3901 56 IM
WALKER, JACK O, KU MED CENTER,66103
588-6522
22 M 1902 53 FP
WALKER, MAURICE A, 3214 STRONG AVE,66106
831-1433
04 M 1601 28 GS
WAXMAN, DAVID, K U MED CENTER,66103
588-1207
18 M 3515 50 IM
WEIGEL, JOHN W, UROLOGY DEPT KU MED CTR,66103
588-6148
29 M 1902 54 U
WELLER, ELIZABETH B, KU MED CENTER,66103
588-6464
49 F 60501 75 P
WELLER, RONALD ALAN, KU MED CENTER - PSY DEPT,66103
588-6464
48 M 2834 74 P

WEST, C OMER, 414 HURON BLDG,66101
371-3572
92 M 1902 23 O
WILLIAMS JR, STERLING B, K U MEDICAL CENTER,66103
588-6200
41 M 401 73 OBG
WILLIAMSON, JOHN A, K U MED CENTER BOX 783,66103
588-5000
51 M 1902 76 ORS
WINN, RANDALL S, KU MED CENTER - RADIOLOGY,66103
588-6800
51 M 1902 76 OR
WINTER JR, CALVERT J, 132 S 17TH,66102
371-2561
22 M 1902 47 PD
WISE III, JOSEPH EDWARD, 132 S 17TH,66102
371-2561
51 M 1902 76 PED
WISNER JR, JOHN HENRY, 229 S EIGHTH,66101
588-6431
46 M 1902 76 P
WOLF, KARL T, 621 NORTHRUP AVE,66101
14 M 1902 48 OO
WONG, BERT Y, K U MED CENTER,66103
588-6015
40 M 801 65 CO
WRIGHT JR, ROBERT W, 669 NEW BROTHERHOOD BLDG,66101
371-5344
24 M 1902 48 GS
YULICH, JOHN O, 1428 S 32ND,66106
384-1630
33 M 1902 59 FP
ZIEGLER, DEWEY K, KU MED CENTER,66103
588-6985
20 M 2401 45 N
ZINN, THOMAS W, 155 S 18TH,66102
371-4343
41 M 1902 67 R

KANSAS CITY, MO—816

ALLEN, MARK LYNN, 3720 WYOMING SUITE 3,64111
53 M 1902
BOUDET, ROBERT A, V A HOSP,64128
861-4700
36 M 1103 62 GS
BROTHERS, MARY ELIZABETH, 2727 MAIN,64108
49 F 1902 74 GS
CALKINS, W GRAHAM, V A HOSP,64128
861-4700
26 M 2501 50 GE
CORDELL, LARRY D, 6400 PROSPECT, 64132
41 M 1902 67 ORS
COULTER, THOMAS B, 4620 J C NICHOLS PKWY,64112
931-0833
38 M 1203 64 OPH
CRIST, ROBERT D, 4400 BROADWAY #416, 64111
561-4400
36 M 1902 63 OBG
CRONEMEYER, RICHARD L, 212 WEST 78TH TERR,64114
52 M 1902
DAVIS JR, JAMES W, V A HOSP,64128
861-4700
33 M 4705 57 IM
DIEHL, ANTONI M, 618 MEDICAL PLAZA,64111
753-4414
24 M 2604 47 POC
ESTES, NORMAN CLEON, 2929 BALTIMORE #250,64108
931-9393
40 M 1902 71 GS
FESTOFF, BARRY W, VA HOSPITAL,64128
861-4700
40 M 1102 77 N
GODFREY, ROBERT G, VA HOSP,64128
861-4700
27 M 1902 58 RHU
HARD, BENJAMIN F, BOX 4913 HAWTHORN RD,64120
242-2525
28 M 4802 55 OBG
JOHNS JR, LEO E, V A HOSP,64128
861-4700
22 M 2101 45 PUD
LEWIS JR, H DANIEL, V A HOSP,64128
861-4700
32 M 5101 58 CO
LUETJE, CHARLES MARION, 2928 MAIN,64108
561-4423
41 M 2803 67 DTO

PAYNE, J RALPH, 4450 ROCKHILL TERRACE,64110
334-2500
40 M 1902 62 EM
PRIETO, JORGE N, 6025 PROSPECT SUITE 102,64132
333-3030
45 M 26401 69 GS
ROLLINS, DOUGLAS E, V A HOSPITAL,64128
861-4700
44 M 4901 73 IM
SINGER, PHILIP A, V A HOSPITAL,64128
861-4700
42 M 3545 69 N
SLEEPER, CAROL A, 20 E FOURTEENTH,64142
-
33 F 1902 58 PATH
STEIN, ROBERT THOMAS, 1010 CARDNOELET DR,64114
-
44 M 2834 70 PD
STILLIE, DO, G DONALD, 7609 LOCUST,64131
-
48 M 2878 78 RT
STOKES, ROBERT LEE, 924 W 34TH,64111
281-8882
43 M 4804 74 EM
VEMIREDDI, NANDA KUMAR, REHABILITATION MED SERVIC,64015
861-4700
41 M 64 PM

KINGMAN—316
(Pratt-Kingman Society)

BLOOM, L THEIL, 708 N MAIN,67068
532-3147
32 M 1902 57 R
BOYER, ROBERT E, 750 AVENUE D WEST,67068
532-5145
36 M 1902 63 FP

KINSLEY—316
(Edwards County Society)

ATWOOD, M DALE, 615 NILES,67547
659-2114
19 M 1902 51 FP
MCKIM, W LYNN, 109 WEST 8TH,67547
659-2137
33 M 1902 59 FP
SCHNDEBELEN, RENE E, 807 EAST 4TH,67547
659-2141
16 M 3901 40 FP

KIOWA—316
(Tri-County Society)

CHRISTENSEN, MARION D, 802 DRUMM,67070
825-4121
25 M 3901 52 FP

LA CROSSE—913
(Central Kansas Society)

BHARGAVA, ASHOK KUMAR, 208 FAIRWAY DR,67548
653-2185
37 M 49547 64 FP

LANSING—913
(Leavenworth County Society)

SNOW, DONALD L, HOLIDAY PLAZA,66043
727-3204
21 M 64901 54 ORG

LARNED—316
(Pawnee County Society)

BRENNER, WILLIAM R, 804 CARROLL,67550
285-3133
15 M 3006 39 FP
CRAM JR, OLE R, 722 MANN,67550
285-2141
18 M 1902 43 FP
DAVIS, DAVID H, 815 W 6TH,67550
285-2131
04 M 1902 30 P
EWING, THOMAS D, 804 CARROLL,67550
285-3133
22 M 1902 45 FP
PARAMESH, JAYA, LSH BOX 89,67550
285-2131
32 F 49608 55 GP
SHAH, MIAN, 313 W 14TH,67550
285-3173
32 M 70403 58 GS
SHAH, NASREEN, 313 W 14TH,67550
285-3173
39 F 70409 62 OBG
SHEPARD, LEROY W, 603 W 5TH,67550
-
04 M 3006 30 OD
SMITH, JOHN O, 804 CARROLL,67550
285-3133
22 M 3901 51 FP

LAWRENCE—913
(Douglas County Society)

ANDERSON, WINSTAN L, 3603 W 10TH ST,66044
-
09 M 1902 34 OD
AUCHARD, VIRGIL M, 2126 LOUISIANA,66044
-
89 M 1902 23 OD
BAILEY, WILLIAM A, PO BOX 127,66044
843-9125
40 M 1902 66 ORS
BELOT JR, MONTI L, LAWRENCE NATIONAL BK BLDG,66044
843-6340
13 M 1902 40 FP
BIERI, PETER V, 1112 W SIXTH ST,66044
841-5217
45 M 1902 71 ENT
BISHOP, RODNEY LEE, 346 MAINE,66044
842-7200
49 M 1902 75 IM
BITTENBENDER, LEE R, 930 IOWA,66044
842-7001
46 M 1902 72 O
BLAIR, T RICHARD, 404 MAINE STE 3,66044
842-3635
34 M 1902 60 IM
BOYDEN, MARY S, MEDICAL ARTS BLDG,66044
842-3778
14 F 2604 38 PDA
BRANSON, VERNON L, 346 MAINE,66044
842-4477
17 M 1902 42 PD
BUCK JR, HENRY W, 1112 W SIXTH,66044
843-0677
34 M 1902 60 OBG
BUTTON, JESSE H, 935 IOWA ST,66044
841-3535
35 M 1902 63 P
CARNAHAN, ROBERT L, 944 KENTUCKY,66044
841-4310
42 M 1902 70 IM
CHEDIAK, ELIAS, 601 MISSOURI,66044
841-7430
39 M 84704 65 P
CLINTON, DALE L, 15 E 7TH,66044
841-5715
21 M 1902 54 PH
CULVER, WARREN T, 2500 W 6TH,66044
842-4178
20 M 3508 46 OPH
DUNLAP, RICHARD L, MEDICAL ARTS CENTER,66044
842-4344
12 M 3005 37 ENT
FRIESEN, DALE, 2500 WEST 6TH,66044
842-7026
47 M 1902 74 ANES
GILLES, HELEN M, MED ARTS CENTER,66044
842-4477
22 F 1902 45 PD
GOODWIN, PHILLIP A, 500 ROCKLEDGE,66044
841-6540
28 M 1902 55 ANES
GRAY, CAPTAIN KING, MEDICAL ARTS BLDG - STE 3,66044
842-7200
48 M 1902 75 IM
HAGGAN, MARGARET E, WATKINS MEM HOSP,66044
843-4455
F FP
HASSELLE III, JAMES E, BERT NASH COMM MTL HLTH,66044
843-9192
35 M 4706 59 P
HATTON, DONALD W, 404 MAINE ST,66044
842-3635
42 M 1902 68 IM
HERMES, RICHARD L, THE MEDICAL ARTS CENTER,66044
843-0677
15 M 4112 39 OBG
HIEBERT, DAVID L, 1112 W SIXTH,66044
841-3211
36 M 1902 61 R
HIRD, WAYNE E, 645 COUNTRY CLUB TERR,66044
842-4300
26 M 1902 50 TS
HUGHES, ROBERT WALTER, MEDICAL ARTS BLDG,66044
843-1374
27 M 1902 54 FP

INGHAM JR, H LAIRD, 404 MAINE,66044
842-3635
45 M 3901 70 IM
JONES, H PENFIELD, MED ARTS CENTER,66044
842-0211
06 M 2401 31 GS
JOSEPH, HOWARD F, DOCTORS BUILDING,66044
843-3981
26 M 1902 51 U
LEARNED, GEORGE R, 401 ARKANSAS,66044
843-5502
22 M 1902 55 GS
LESSENDEN, GLENN A, MED ARTS BLDG,66044
843-6233
24 M 1902 48 FP
LOVELAND, G CHARLES, 346 MAINE,66044
842-4477
47 M 1902 73 PD
MADSEN, GLENN L, 1112 W SIXTH,66044
841-3211
38 M 3005 65 R
MANAHAN, G EUGENE, MED ARTS CENTER,66044
842-0211
19 M 1902 44 GS
MITCHELL, ALEX C, 1626 W 20TH,66044
843-4739
18 M 1902 50 FP
MOORELL, CAROL A, 404 MAINE,66044
843-3680
45 F 1902 71 PATH
MONCKTON, LAWRENCE A, 1112 W SIXTH SUITE 204,66044
843-2010
48 M 1902 74 GS
NELSON, RICHARD O, 935 IOWA,66044
843-0921
11 M 1001 41 FP
OELSCHLAGER, RONALD O, 1112 W SIXTH,66044
841-3211
43 M 1902 69 R
OLSON, CARL E, 935 IOWA,66044
842-9911
17 M 1611 46 FP
ORCHARD, RICHARD A, 1112 W SIXTH SUITE 212,66044
841-2280
41 M 2802 68 OPH
OWEN, GARRY O, 1112 W SIXTH,66044
843-0677
37 M 1902 63 OBG
PEES JR, GERALD BOYD, 901 KENTUCKY,66044
843-5160
45 M 1902 71 IM
PRAEGER, MARK A, 1112 W SIXTH SUITE 204,66044
843-2010
42 M 1902 68 GS
PRICE JR, LAURANCE W, C/O LAWRENCE CLINICAL LAB,66044
843-3680
33 M 1902 59 PATH
REED, RALPH R, 404 MAINE,65044
842-3635
27 M 1902 53 IM
REESE, JOHN L, 346 MAINE,66044
842-6644
35 M 1902 61 GS
ROBERTS, RICHARD S, 308 MAINE,66044
843-6137
19 M 2802 44 GS
SANDERS, J ALAN, LAWRENCE MEMORIAL HOSP,66044
842-2083
29 M 1902 60 PATH
SCHNOSE, GREGORY O, 901 KENTUCKY #101,66044
843-5160
51 M 1902 76 IM
SCHROEDER, SYDNEY O, WATKINS MEMORIAL HOSP,66044
864-4045
18 M 1902 44 P
SCHWEGLER, RAYMOND A, MEDICAL ARTS CENTER,66044
843-4455
07 M 2604 31 OBG
TUCKER, DONALD R, 2412 UNIVERSITY DR,66044
354-5100
31 M 1902 57 IM
TUCKER, VIRGINIA L, 2412 UNIVERSITY DR,66044
843-3750
30 F 1902 57 OD
WALTERS, BYRON W, 1030 AVALON RD,66044
843-6751
13 M 1902 39 FP
WELL, MICHAEL A, 944 KENTUCKY,66044
843-3981
41 M 1606 67 U
WERTZBERGER, JOHN, PO BOX 127,66044
843-9125
36 M 1902 63 ORS
WERTZBERGER, KENNETH LYNN, 1211 W SIXTH,66044
843-9125
47 M 1902 73 ORS
WILCOX SR, HOWARD L, MEDICAL ARTS CENTER,66044
843-0677
18 M 3520 44 OBG
WOLLMANN, MARTIN, 2615 ORCHARD LN,66044
843-9530
26 M 1902 57 IM
WRIGHT, M ERIK, 2728 STRATFORD ROAD,66044
-
15 M 502 50 P

LEAVENWORTH—913
(Leavenworth County Society)

AL-BAGHAL, MOHAMMAD, 520 SIXTH AVE,66048
682-6661
45 M 87501 72 U
ANWAR, M ZIA, 109 DELAWARE,66048
651-2977
39 M 11801 67 IM
BARRY, DAVID R, 500 EISENHOWER RD,66048
727-6000
42 M 1902 68 FP
BENNETT, CHARLES A, 1009 S BROADWAY,66048
-
96 M 1902 25 OO
CENAC, MARK T, 500 EISENHOWER RD,66048
727-6000
27 M 401 49 GS
COMBS, G RALPH, 510 FIFTH,66048
-
79 M 4101 02 OO
COMBS, PETER S, 213 DELAWARE,66048
682-0242
14 M 4101 41 IM
DE SOUZA, DERRICK J, 520 SIXTH AVE,66048
682-6661
43 M 49501 66 GS
OUYSAK, SAMI, 520 5TH AVE,66048
682-6661
22 M 90201 47 IM
GERBER, HARRY A, 605 N 6TH,66048
682-3893
96 M 5606 30 FP
GRAHAM, J MALCOLM, 500 EISENHOWER RD,66048
727-6000
28 M 3840 54 PD
GRAHAM, KENNETH L, 500 EISENHOWER RD,66048
727-6000
21 M 3840 45 GS
GRAHAM, THOMAS W, 500 EISENHOWER RD,66048
727-6000
26 M 3840 50 IM
GRISOLIA, ANDRES, 424 WALNUT ST,66048
682-5400
27 M 84708 50 ORS
HAMMEKE, JOHN C, 3601 S 4TH ST TRAFFICWAY,66048
682-5201
27 M 401 61 OPH
JOHLER, TERRY HARTWIG, 500 EISENHOWER RD,66048
727-6000
40 M 2802 67 FP
JOHNSON, PAUL D, 520 SIXTH AVE,66048
682-6661
36 M 1902 61 FP
MCCOLLUM, MARY B, 607 SHAWNEE,66048
651-6566
43 F 4804 68 P
MCCOLLUM, WILLIAM B, 3601 S FOURTH,66048
682-6661
41 M 1902 66 TS
MCKEE, RICHARD S, 1210 S BROADWAY,66048
682-1023
05 M 1902 35 ANES
MERRITT, W HENRY, 520 SIXTH AVE,66048
682-6661
14 M 702 39 GS
NAGESH, KAVI G, PO BOX 1204,66048
682-2000
32 M 49509 58 IM
PARKER, ROBERT W, 500 EISENHOWER RD,66048
727-6000
45 M 1902 71 FP
RABE, MELVIN A, 520 SIXTH AVE,66048
682-6661
14 M 1902 37 GS
STRUTZ, WILLIAM C, 68 WESTWOOD DR,66043
682-8868
08 M 5605 43 R
TAMBURINI, MARIO, 2605 S 16TH,66048
682-8950
16 M 13201 49 R

VOORHEES, CARROLL D, 520 SIXTH AVE,66048
682-6661
2S M 1902 52 FP
VOORHEES, GORDON S, 520 SIXTH AVE,66048
642-6661
12 M 1902 39 IM

LEBO—316
(Coffee Flint Hills Society)

HUNTER, KENNETH R, ,66856
256-2565
07 M 1902 39 FP

LENORA—913
(Northwest Kansas Society)

STEICHEN, EDWARD F, ,67645
05 M 1601 31 OO

LIBERAL—316
(Seward County Society)

ALLEN, RAY E, 2 PLAZA DR,67901
624-5691
37 M 1902 64 IM
CAEDO, CARMELITA O, PO BOX 1643 SW MED CENTER,67901
624-1651
41 F 74801 63 R
CAMPION, WOODROW M, 121 W THIRD,67901
624-2594
13 M 1902 39 IM
GRIMES, I ROSS, 222 W 15TH,67901
624-1676
27 M 3901 54 TS
HARRIS, NORVAN D, 222 W 15TH,67901
624-3811
20 M 1902 44 OBG
HOLCOMB, WILLIAM M, 15 E 11TH,67901
624-2252
31 M 3901 56 GS
KOONS, JESS W, 1210 N WASHINGTON,67901
624-3841
27 M 1902 57 OPH
NEVINS, RICHARD L, PO BOX 1824,67901
624-1841
47 M 3901 73 FP
PALMER JR, H C, 2 PLAZA DRIVE,67901
624-5691
36 M 1902 63 IM
PROCHAZKA, OTTO F, BOX 1809,67901
624-1651
12 M 1902 38 R
RATHBUN, EDWIN D, 610 W 11TH,67901
624-1841
36 M 1902 62 FP
REESE, JACK D, 15 E 11TH,67901
624-6226
32 M 1902 57 FP
SEAGO, CHARLOTTE L, 1031 N KANSAS,67901
624-1625
35 F 1720 60 PO
WADE, THEODORE E, 318 N LINCOLN,67901
04 M 512 30 OO

LINCOLN—913
(Central Kansas Society)

MADDEN, WILLIAM J, 206 WEST LINCOLN,67455
24 M 1902 51 OO
SONGER, HERBERT L, ,67455
524-4053
12 M 1902 38 FP

LINDSBORG—913
(McPherson County Society)

FREDRICKSON, DUANE E, 121 W LINCOLN,67456
227-3371
39 M 1902 66 FP
FULLER, DERYL D, 121 W LINCOLN,67456
227-3371
25 M 1902 50 FP
MURFITT, MALCOLM C, 231 N MAIN,67456
227-2732
13 M 801 41 FP

LINWOOD—913
(Leavenworth County Society)

FUNK, EDWARD D, BOX 144 A-1,66052
04 M 1902 41 ANES

LYNDON—913
(Franklin County Society)

STOUT, NILES M, ,66451
828-4521
16 M 1902 50 FP

LYONS—316
(Rice County Society)

GRIMES, JAMES T, 1221 W NOBLE,67554
257-5124
27 M 1902 53 FP
HAYES, KRIS A, 201 S PIONEER,67554
257-5971

SIEMENS, RICHARD A, 1221 W NOBLE,67554
257-5124
30 M 1902 59 FP
WOLF, CURTIS V, 1221 W NOBLE,67554
257-5124
37 M 1902 64 FP

MANHATTAN—913
(Riley County Society)

BAKER, RICHARD B, 1133 COLLEGE,66502
537-4200
42 M 4113 68 ORS
BALL, RALPH G, 215 S DELAWARE,66502
03 M 1902 27 OO
BASCOM, GEORGE S, 1133 COLLEGE,66502
539-6574
27 M 2401 52 GS
BOESE, KENNETH M, 1133 COLLEGE AVE,66502
776-4744
25 M 1902 56 FP
BROWN, ROBERT M, 1133 COLLEGE,66502
532-6544
31 M 1902 63 FP
BURDICK, BRUCE M, 117 S FIFTH,66502
776-9411
25 M 512 53 P
CATHEY, ROBERT H, 1133 COLLEGE AVE,66502
537-4990
42 M 1902 68 D
CRANE, C HERBERT, 1133 COLLEGE,66502
537-9030
22 M 3520 46 PO
DURKEE, WILLIAM R, 1133 COLLEGE AVE,66502
776-4744
23 M 1902 45 IM
FAIRCHILD, JOHN A, 756 COLLEGE HTS CIR,66502
539-4041
14 M 3006 41 OO
FISCHER, REX R, 1133 COLLEGE,66502
539-5322
34 M 3005 60 OBG
FREEMAN, FRED A, MANHATTAN MED CENTER,66502
537-8710
42 M 1902 69 U
GARDNER, JAMES D, 1133 COLLEGE AVE,66502
537-4940
43 M 2834 71 IM
HEASTY, ROBERT G, 1133 COLLEGE,66502
539-5322
11 M 3519 38 OBG
HOLT, BEVLEY DAN, PO BOX 1144,66502
26 M 4706 64 R
HOSTETTER, PHILIP H, 821 POYNTZ,66502
537-2544
17 M 1902 42 FP
HUNTER JR, JAMES S, 1133 COLLEGE,66502
539-5501
18 M 2301 41 OBG
JUBELT, HILBERT P, 1133 COLLEGE,66502
537-9030
19 M 1611 43 PO
KALDOR, RICHARD H, PO BOX 128,66502
539-5363
40 M 2401 66 PATH
KEMPTHORNE, CHARLES R, 519 N 11TH,66502
03 M 5605 32 OO
KIRK, THOMAS E, 206 SOUTHWIND,66502
537-0655
44 M 3005 71 OPH
KLINGLER JR, EUGENE A, 1133 COLLEGE,66502
539-5341
35 M 1902 62 GS
LAFENE, BENJAMIN W, 1844 ANDERSON AVE,66502
01 M 3806 31 OO

LOWE, STANLEY W, 1133 COLLEGE AVE,66502
776-3451
32 M 1902 59 OPH
LYONS JR, FRANK C, 1133 COLLEGE AVE,66502
539-7641
44 M 3840 70 DR
MARSHALL, RONALD L, 1133 COLLEGE AVE,66502
539-5322
42 M 3005 67 OBG
MARTIN, DANIEL C, LAFENE HEALTH CENTER KSU,66506
532-6544
30 M 1902 58 IM
MCKNIGHT, DAVID E, 1133 COLLEGE AVE,66502
539-7641
32 M 1902 62 R
MCNEIL, ELBERT D, 1133 COLLEGE,66502
537-9030
22 M 702 48 PD
MEEK, PALMER F, 1133 COLLEGE,66502
537-2651
45 M 1902 71 IM
MILLER, ABRAHAM H, 1133 COLLEGE,66502
537-2651
29 M 4101 54 IM
MOSIER, STEVEN J, 215 SOUTHWIND PLACE,66502
776-9761
49 M 1902 74 FP
MOWRY, GERALD L, 1133 COLLEGE,66502
539-5322
26 M 1902 53 OBG
OLNEY, ROBERT D, 1133 COLLEGE AVE,66502
539-7555
27 M 3005 51 GS
PETERSON, JACK T, 1133 COLLEGE,66502
539-5363
25 M 1902 50 PATH
PHILIPP, JOSEPH THEODORE, 1115 WATERS,66502
537-7373
45 M 1902 67 OPH
PHILLIPS, STEPHEN B, LAFENE ST HEALTH CENTER,66506
532-6544
17 M 1902 45 IM
REITZ, LELAND C, 1133 COLLEGE,66502
537-2651
36 M 1902 63 IM
REITZ, ROGER P, 1133 COLLEGE,66502
537-2651
32 M 1902 59 IM
ROSE, GRAHAM C, 1133 COLLEGE,66502
537-9030
46 M 4705 70 PD
STEIN, ROBERT B, 2312 ANDERSON - SUITE 305,66502
539-1851
29 M 3515 52 P
STONE, G REX, 1133 COLLEGE AVE,66502
539-7555
29 M 1902 54 GS
TIEMANN, WILLIAM H, 1133 COLLEGE,66502
532-6544
42 M 3005 67 FP
VOLKMANN II, HARLEY W, 1133 COLLEGE AVE,66502
539-7641
47 M 1902 72 R
WHITE, THADDEUS H, 1735 ANDERSON AVE,66502
15 M 1902 42 OO

MANKATO—913
(Republic County Society)

KIMBALL, RICHARD R, 102 S CENTER,66956
378-3511
45 M 1001 72 FP
SCHLOTTERBACK, WILLIAM E, 517 N HIGH ST,66956
378-3511
31 M 1902 61 FP

MARION—316
(Marion County Society)

DIAMENT, MORTON LEONARD, 504 S ROOSEVELT,66861
382-3123
29 M 80301 64 FP
ENSEY, T CRANFORD, 504 S ROOSEVELT,66861
382-2182
20 M 4804 47 FP
GUERRA, TOMAS H, 122 S CEDAR,66861
382-2137
33 M 84704 65 IM

MARYSVILLE—913
(Northeast Kansas Society)

ARGO, DONALD A, 808 N 19TH,66508
562-2303
36 M 3005 64 FP
LAWS, LEWIS R, 808 N 19TH,66508
443-3121
25 M 1902 54 FP

McLOUTH—913
(Jefferson County Society)

SNOOK, ROBERT RUFUS, ,66054
796-6116
11 M 1902 42 FP

McPHERSON—316
(McPherson County Society)

BILLINGS, THOMAS, 400 WEST 4TH,67460
241-5500
39 M 1902 66 FP
BRANDSTED, ERNEST C, 400 W 4TH,67460
241-1654
18 M 1606 44 OBG
COLLIER, WILLIAM J, 400 W 4TH,67460
241-1766
25 M 3605 49 TS
DYCK, ARTHUR H, 101 1/2 NORTH MAIN,67460
241-0357
03 M 1902 28 FP
FERREE, RICHARD ALLAN, 400 W FOURTH,67460
241-7400
51 M 3006 76 FP
JOHNSON, J RICHARD, 400 W 4TH,67460
241-4293
28 M 1902 55 IM
OUANO JR, BIBIANO B, MEMORIAL HOSP,67460
241-2250
40 M 74801 63 U
PIERSON, W WEIR, 823 N MAIN,67460
241-1445
17 M 1902 44 FP
PRICE, VAUGHAN C, 120 N ASH,67460
241-1224
05 M 4706 29 GS
SOHLBERG JR, ROBERT, PO BOX 346,67460
05 M 1606 34 FP
THOMAS, GREGORY MCQUEEN, 400 W FOURTH,67460
241-7400
47 M 1902 73 FP

MEADE—316
(Iroquois County Society)

DAUGHERTY, ROBERT M, 631 E WALNUT,67864
09 M 1902 36 FP
DONAHOE, DORVAL H, PO BOX 488,67864
873-2181
32 M 5501 63 FP
HILL, RICHARD H, 234 EAST CARTHAGE,67864
873-2113
18 M 1902 44 FP

MEDICINE LODGE—316
(Tri-County Society)

HOFFER, JOHN G, 910 N WALNUT,67104
886-3222
13 M 1902 44 GS
STUCKY, DEAN E, 116 E KANSAS,67104
886-5653
33 M 1902 60 FP

MINNEAPOLIS—913
(Saline County Society)

BARKER, STEVEN E, 311 N MILL,67467
392-2144
51 M 1902 76 FP
HUNNINGHAKE, RONALD E, 311 N MILL,67467
392-2144
51 M 1902 76 FP
WEDEL, KENNETH D, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP
WEDEL, KERMIT G, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP

MINNEOLA—316
(Iroquois County Society)

STEPHENS, CHARLES, MINNEOLA CLINIC,67865
885-4202
33 M 2803 58 FP
TROTTER, ROGER COURTNEY, MINNEOLA CLINIC,67865
885-4202
47 M 1902 74 FP

MONTEZUMA—316
(Ford County Society)

PHAM, TOAI KE, MONTEZUMA CLINIC,67867
846-2251
34 M 94101 62 GP

MORAN—316
(Allen County Society)

NEVITT, J RUSSELL, P O BOX 430,66755
–
99 M 3510 32 OO

MOUNDRIDGE—316
(McPherson County Society)

KAUFMAN, WILLARD E, 115 N CHRISTIAN AVE,67107
345-6322
28 M 1902 53 FP
LOGANBILL, VAROEN J, 115 N CHRISTIAN AVE,67107
345-6322
26 M 1902 54 FP

MULVANE—316
(Sedgwick County Society)

COBB, LESLIE H, 102 E MAIN,67110
777-1441
17 M 4804 47 FP

NASHVILLE—316
(Pratt-Kingman Society)

WAYLAN, THORNTON L, ,67112
–
06 M 1902 35 OO

NEODESHA—316
(Southeast Kansas Society)

CHRONISTER, BERT, PO BOX 118,66757
325-2622
38 M 1902 64 FP
MOORHEAD JR, F ALLEN, 709 MAIN ST,66757
325-2200
39 M 1902 65 FP

NESS CITY—913
(Central Kansas Society)

PRAKALAPAKORN, DARANEE, 412 N TOPEKA,67560
798-2233
47 F 89101 69 PO
PRAKALAPAKORN, YANYONG, 412 N TOPEKA,67560
798-2233
43 M 89101 69 GS
WIENS, PETER K, BOX B,67560
798-3191
24 M 1902 55 FP

NEWTON—316
(Harvey County Society)

ALLEN, FRANCES A, 1112 BOYD,67114
–
15 F 1902 43 OO
BATES, MICHAEL NICHOLS, 301 MAIN,67114
283-7257
50 M 1902 75 OBG
BENTON, JAY S, 301 MAIN,67114
283-3600
23 M 4804 49 OBG
CAMPBELL, FRANCES S, 1901 E FIRST,67114
283-2400
35 F 4101 61 P
CARPER, IVAN H, AXTELL CLINIC,67114
283-2800
28 M 1902 59 GS
CARPER, OWEN E, AXTELL CLINIC,67114
283-2800
37 M 1902 64 FP
CLAASSEN, MILTON A, 201 S PINE ST,67114
283-3600
32 M 1902 58 ORS
CRAIG, CHARLES C, 203 E BROADWAY,67114
283-2800
45 M 1902 71 ORS
CRANSTON, STEPHEN O, AXTEL CLINIC,67114
283-2800
71 M 1902 71 GS
DYCK, GEORGE, PRAIRIE VIEW INC,67114
268-8388
37 M 6201 64 P

ENNS, EUGENE K, 6 INDIAN LANE,67114
–
15 M 1902 40 OO
ENNS, JAMES H, 900 N POPLAR,67114
283-1400
24 M 1902 47 OPH
FENT, LEE S, 316 OAK,67114
283-0505
14 M 2834 43 GS
FRANSEN, HERBERT, 209 S PINE,67114
283-5040
32 M 6501 60 GS
FRANSEN, PAUL H, 209 S PINE,67114
283-5040
46 M 6501 71 FP
GLOVER, RICHARD M, 203 E BROADWAY,67114
283-2800
21 M 1902 53 FP
GRISWOLD, DALE G, 203 E BROADWAY,67114
283-2800
27 M 1902 53 IM
GROVE, JOHN A, AXTELL CLINIC,67114
283-2800
08 M 1606 37 ORS
HWA, EUGENE C, 500 MAIN,67114
283-1160
21 M 24216 47 R
ISAAC, CHARLES A, AXTELL CLINIC,67114
283-2800
25 M 1902 49 U
KLASSEN, DANIEL S, 316 OAK,67114
283-1313
13 M 1902 42 OBG
KLIEWER, VERNON L, PRAIRIE VIEW MHC,67114
283-2400
31 M 1605 57 CP
KUMAR, SURINDER, 201 S PINE,67114
835-2241
46 M 1902 69 OBG
MYERS, ROBERT W, RT 1,67114
–
11 M 1902 43 OO
NACHTIGALL, ANDREW, BETHEL CLINIC,67114
283-3600
28 M 1902 59 PD
O'TOOLE, JAMES K, PRAIRIE VIEW HOSPITAL,67114
283-2400
28 M 1643 54 P
OLSON, ERWIN T, BETHEL CLINIC,67114
283-3600
19 M 1902 47 PD
PEORAZA, HERNANDO V, 4 SYCAMORE CT,67114
264-1381
28 M 26404 56 R
PREHEIM, DELBERT V, 209 S PINE,67114
283-5040
13 M 702 42 IM
QUAMAR, YUSUF, AXTELL CLINIC,67114
283-2800
38 M 70409 62 IM
RADOVANOV, RADMILA, 500 MAIN,67114
–
34 F 95702 60 R
RICH, ELDON S, BETHEL CLINIC,67114
283-6000
16 M 1902 46 GYN
SCHMIDT, HERBERT R, CEDAR VILLAGE,67114
–
03 M 1902 34 OO
SILLS, CHARLES T, 1631 HILLCREST,67114
283-0177
09 M 1902 37 IM
SIMMONS, ROBERT EARLE, 209 S PINE,67114
283-5040
49 M 1902 74 IM
TANDOC JR, VALENTIN T, BETHEL CLINIC,67114
283-3600
39 M 74809 62 U
TOMPKINS, CARL O, 316 OAK STREET,67114
283-1380
22 M 1902 51 FP
VAUGHAN, DONNA A, 201 S PINE,67114
283-3600
45 F 1902 71 IM
VOGT, VERNON W, BETHEL CLINIC,67114
283-3600
22 M 3005 53 FP
VON GRASEMSIRI, SUNAN, 500 NORTH MAIN,67114
283-1160
41 M 89101 67 R
WEBER, ROY R, 209 S PINE,67114
283-5040
46 M 1902 73 IM

WHEELER, DWIGHT E, BETHEL CLINIC,67114
283-3600
50 M 76 1M
WIENS, J WENDELL, 201 S PINE,67114
283-3600
32 M 1902 59 GS

NORTON—913
(Northwest Kansas Society

COLIP, F MERLYNN, 711 N NORTON,67654
927-3305
35 M 1902 61 FP
COOPER, ARTHUR E, 305 W WILBERFORCE,67654
—
08 M 1611 34 GP
HARTLEY, ROY WILEY, 711 N NORTON,67654
927-3305
37 M 1902 63 FP
HARTMAN, ROGER L, 711 N NORTON,67654
927-3305
35 M 1902 61 FP
LONG, ROBERT C, 711 N NORTON,67654
927-3305
27 M 1902 53 GS

NORTONVILLE—913
(Jefferson County Society)

MADISON, WILLARD A, ,66060
886-2110
20 M 1902 51 FP

OAKLEY—913
(Northwest Kansas Society)

OHMART, RICHARD V, PO BOX 756,67748
672-3262
36 M 1902 62 FP
SEKAVEC, GORDON B, 209 CENTER AVE,67748
672-3351
07 M 1902 38 FP

OBERLIN—913
(Northwest Kansas Society)

SIMPSON, ROBERT LIMBAUGH, OBERLIN CL,67749
475-2221
25 M 4706 51 GS
WHITAKER, REN R, 902 W COLUMBIA,67749
475-2222
37 M S404 66 FP

OLATHE—913
(Johnson County Society)

ARONOFF, MICHAEL E, 407 S CLAIRBORNE STE 209,66061
782-3953
39 M 1604 64 ENT
BEEBE, EDMER, 420 EAST CEDAR,66061
—
03 M S605 32 FP
BLISS, JOY V, 42 HOLLY DRIVE,66061
782-2292
42 F 3005 68 ANES
BOSWELL, H CRAIG, 1502 LAKESTONE DR,66061
588-6670
47 M 1803 73 ANES
BROWN, PAUL W, 405 S CLAIRBORNE,66061
782-3322
44 M 1902 70 FP
CHENOWETH, JOHN R, D.O., 304 S CLAIRBORNE,66061
782-6100
36 M 2878 70 OST
DELPHIA, ROBERT E, 401 S CLAIRBORNE,66061
782-1610
24 M 1902 56 FP
DOUGLAS, JOSEPH MAHLO, JOHNSON CO MENTAL HLTH CT,66061
782-2100
39 M 1902 65 P
EIDT, DAVID W, 407 S CLAIRBORNE,66061
782-8487
44 M 2501 70 FP
EIDT, LAURENCE A, 407 SOUTH CLAIRBORNE,66062
782-8487
44 M 1902 71 FP
FORTUNE, CEDRIC B, 405 S CLAIRBORNE,66061
782-3322
40 M 1902 66 FP
GLAZZARD, CHARLES D, 407 S CLAIRBORNE,66061
782-3384
28 M 2507 56 P
HALVORSON, HOWARD C, 407 S CLAIRBORNE,66061
782-2020
41 M 5404 66 U
HUDSON, ROBERT P, 12925 FRONTIER RD,66061
588-7040
26 M 1902 52 1M
JENSEN, THOMAS M, 407 S CLAIRBORNE,66061
782-1148
47 M 3005 73 ORS
LAIRD, DALE D, ONE PATRONS PLAZA,66061
782-3631
42 M 1902 68 OPH
MATTHEW, WILLIAM L, 405 S CLAIRBORNE,66061
782-3322
29 M 1902 56 FP
MCCANN, WILLIAM E, 540 E SANTA FE,66061
782-0262
22 M 3901 48 FP
MEE, ADRIAN W, 28 HOLLY DRIVE,66061
782-2292
19 M 1902 54 ANES
MELENDRES, JUANITO M, 7400 GLEASON, RR1, 66061
—
36 M 74809 61 PD
MILLIGAN, DONALD B, 401 S CLAIRBORNE,66061
782-1610
48 M 2307 74 FP
MORGAN II, DAVID LLOYD, 807 S CLAIRBORNE,66061
782-8300
49 M 2820 77 1M
PIERRON, GEORGE J, 540 EAST SANTA FE,66061
782-0260
22 M 1902 47 FP
PINCOMB, ARTHUR L, 401 S CLAIRBORNE,66061
782-1415
20 M 1902 51 FP
ROMONDO, STEVEN A, 300 SOUTH ROGERS RD,66061
782-2292
47 M 1902 73 ANES
RUHLEN, JAMES L, 807 S CLAIRBORNE,66061
782-8300
46 M 1902 72 1M
SEAMAN, LAUREN I, 401 CLAIRBORNE,66061
782-1415
07 M 6001 38 FP
SETTLE JR, RUSSELL O, 407 S CLAIRBORNE,66061
782-3384
35 M 1902 60 P
SHEFFER, KEITH D, 407 S CLAIRBORNE STE 101,66061
782-1148
37 M 1720 67 ORS
SNYDER JR, RICHARD HENRY, 300 S ROGERS ROAD,66061
782-1451
45 M 1902 73 ANES
YEOMANS, RONALD N, 405 S CLAIRBORNE,66061
782-3073
40 M 1902 67 OBG

ONAGA—913
(Pottawatomie County Society)

FLECKENSTEIN, CHARLES S, 501 LUCIEN ST,66521
889-4601
07 M 1902 36 OO
WALSH, THOMAS E, ONAGA CLINIC,66521
889-4241
48 M 1902 74 FP

OSAGE CITY—913
(Flint Hills Society)

WILLIAMS, HOMER J, 611 S SIXTH,66523
—
05 M 1902 31 FP

OSAWATOMIE—913
(Miami County Society)

APPENFELLER, WILLIAM O, 524 BROWN AVE,66064
755-3166
25 M 1902 53 FP
MORALES, AMALIA D, P O BOX 500,66064
755-3151
22 F 21501 50 AOM
MORALES, OTTO E, P O BOX 500,66064
755-3151
22 M 27501 51 P
QUINONES, ELADIO A, OSAWATOMIE STATE HOSP,66064
755-3151
16 M 27501 43 FP

OSWEGO—316
(Labette County Society)

BURGESS, ARTHUR P, 504 5TH STREET,67356
795-4427
19 M 1902 52 FP

SHORTES, LOIS E, 220 UNION,67366
795-4868
FP

OTTAWA—913
(Franklin County Society)

BLANKENSHIP, JIM O, 1320 S ASH,66067
242-1620
50 M 2834 76 FP
CORDER, S SCOTT, 1502 S CEDAR,66067
242-2641
51 M 1902 76 FP
GOLLIER, ROBERT A, MED ARTS BLDG,66067
-
13 M 1902 37 OO
GOLLIER II, ROBERT A, MEDICAL ARTS BLDG,66067
242-1620
40 M 1902 66 FP
HADLEY, DELMONT C, 1320 SOUTH ASH,66067
242-3891
35 M 1902 64 FP
HENNING, CALVIN W, 1502 CEDAR,66067
242-2641
05 M 1902 35 FP
LAURY, DAVID G, 1320 S ASH,66067
242-1620
17 M 1606 44 FP
LOFGREEN, VICTOR J, ROUTE #1,66067
-
97 M 1902 32 OO
PHILGREEN, DONALD E, 1320 S ASH,66067
242-3891
39 M 1602 67 FP
REYES JR, FRANCISCO A, 1320 S ASH,66067
242-5312
38 M 74801 61 GS
SAYLOR, STEPHEN, 1320 ASH,66067
242-1620
47 M 1902 73 FP
SPEER, LOUIS N, PO BOX 0,66067
242-1257
14 M 1605 41 FP
STREHLOW, CHESTER H, MEDICAL ARTS BUILDING,66067
242-3891
30 M 1902 57 FP

OVERBROOK—913
(Shawnee County Society)

RUBLE JR, JAMES L, OVERBROOK COMM CLINIC,66524
665-2205
26 M 1902 53 FP

PAOLA—913
(Miami County Society)

BANKS, ROBERT E, PO BOX 298,66071
294-2305
29 M 1902 55 FP
NAIK, GOPAL V, 700 BAPTISTE DR,66071
294-5316
46 M 49501 71 OBG
ROWLETT, JACK G, PO DRAWER A,66071
294-2356
21 M 1902 52 FP
STANLEY, REX C, PO DRAWER A,66071
294-2356
24 M 1902 52 GS

PARSONS—316
(Labette County Society)

AVES, AGNES, 1509 MAIN,67357
421-0600
38 F 74801 59 IM
AVES, RENATO B, 1509 MAIN STREET,67357
421-0600
35 M 74801 59 GS
BORKLUND, MAURICE K, 400 KATY,67357
421-2700
21 M 1720 50 GS
CRAMER, GUY W, 412 MURDOCK,67357
-
11 M 1902 39 OO
DE BRIERE, SIDNEY L, BOX 738,67357
-
19 M 702 52 OO
HENDERSON, CHARLES F, 1509 MAIN,67357
421-0600
14 M 1902 40 A
LAVA, CHIRUND, 107 MAIN PO BOX 290,67357
421-6210
40 M 89102 63 GS
LIUJULUSCHAN, RIT, 107 MAIN,67357
421-6210
41 M 89101 68 GS
MARTIN, EARL A, 1516 GRAND,67357
-
07 M 1606 35 OO
MILLER, CHARLES H, 2819 CLARK,67357
-
07 M 3006 32 OO
MILLER, DEAN M, 203 CRESTVIEW,67357
421-4880
22 M 1902 48 R
MILLER, STEPHEN FRANCIS, 1509 MAIN,67357
421-0600
45 M 1902 70 GS
PACE, JOHN D, KATY HOSPITAL CLINIC,67357
421-1010
94 M 1902 20 FP
PAI, RADHA V, 1710 CORNING,67357
421-0080
45 ANES
PAI, VARADARAJ S, 1710 CORNING,67357
421-0080
42 U
PARANJOTHI, SUBRAMONIAM P, 1509 MAIN,67357
421-6160
39 M 49531 64 IM
PAULS, DANIEL N, 2600 CORNING,67357
421-1431
45 M 1902 71 IM
ROJAN, CHAVALIT, 310 N 16TH,67357
421-2460
47 M 89102 72 PD
ROTHSTEIN, TERRY B, 220 NORTH 32ND,67357
421-5900
43 M 1606 69 OPH
SWARTZ, WARREN E, 400 KATY AVE,67357
421-2700
25 M 1902 52 GS
TANGCHUPONG, CHANTRASIRI, 310 N 16TH,67357
421-2460
47 F 89102 71 PD
TANGCHUPONG, SAROHO, 310 N 16TH,67357
421-2460
43 M 89102 69 OBG
VERMA, ASHA, 400 KATY,67357
421-9090
37 F 49530 63 PD
WHITE, JOHN P, PARSONS CLINIC,67357
421-0600
17 M 1902 42 FP

PHILLIPSBURG—913
(Central Kansas Society)

RICHARDS, JON F, PHILLIPS COUNTY MED CT,67661
543-5211
50 M 1902 75 FP

PITTSBURG—316
(Crawford County Society)

ARMSTRONG, HAROLD J, PROFESSIONAL BUILDING,66762
232-2600
40 M 1902 68 ORS
BENA, JAMES H, 109 EAST 9TH,66762
231-6950
12 M 3005 36 PD
BERKEY, VERNON A, KIRKWOOD BLDG,66762
231-7650
18 M 1902 43 R
BIERLEIN, KENNETH J, 812 S CATALPA,66762
-
06 M 1606 33 OO
CARSON, RICHARD CARLYLE, MT CARMEL HOSPITAL,66762
231-6100
35 M 1720 63 ANES
COOMER, TYLER E, 315 NATL BANK BLDG,66762
231-7730
30 M 2101 59 GS
COOPER, KENT J, 909 CENTENNIAL,66762
231-6280
41 M 1902 73 FP
ERICKSON, CLARENCE W, 217 NATL BANK BUILDING,66762
231-7400
06 M 1902 33 IM
ESCH, JOHN G, 207 KIRKWOOD BLDG,66762
231-5360
24 M 3006 48 GS
GOMEZ, MODESTO S, 909 E CENTENIAL,66762
231-2490
35 M 72601 63 PD
HOLSINGER, DONALD M, 1015 MT CARMEL PL,66762
231-5900
38 M 1902 64 IM

HUEBNER, ROBERT STEPHAN, NATIONAL BANK BLDG,66762
231-6160
42 M 1606 67 GS
HUERTER, DAVID F, 909 CENTENNIAL,66762
231-1650
46 M 1902 72 IM
LANCE, RAYMOND W, 608 W QUINCY,66762
-
22 M 1902 47 OO
LEFFLER, PAUL B, 309 WINWOOD,66762
-
02 M 1902 40 OO
MCKEE, DICK B, 1500 S COLLEGE,66762
231-6220
96 M 1902 28 FP
MILLER, EARL E, 1312 S BROADWAY,66762
231-6410
13 M 1902 37 ENT
MULLER, SAMUEL B, 511 W QUINCY,66762
-
05 M 1902 34 OO
NEWMAN, CLIFFORD B, 1204 E 7TH,66762
-
01 M 1902 28 OO
ODGERS, RODNEY K, 909 CENTENNIAL,66762
231-4300
M 1902 74 IM
PARSI, MANUTCHEHR K, 909 CENTENNIAL,66762
231-3770
38 M 51701 64 OBG
POGSON, GEORGE W, 1015 MT CARMEL PLACE,66762
231-5900
24 M 1902 47 IM
RAMIREZ, AUGUSTO H, 909 CENTENNIAL,66762
231-1600
32 M 26407 58 IM
RAMIREZ, IRENE, 909 CENTENNIAL,66762
231-6280
F
SCHLEMMER, ROGER B, 1009 S BROADWAY,66762
231-6380
37 M 1902 68 OPH
SEGLIE, FLOYD RONALD, 909 CENTENNIAL DR SUITE 3,66762
231-6280
43 M 1902 69 FP
TWEET, FREDRICK A, MT CARMEL HOSPITAL,66762
231-6100
39 M 1602 66 PATH
WOOD, DOUGLAS H, 413 W JEFFERSON,66762
-
11 M 5605 36 OO
YAGHMOUR, TALAAT E, 905 NORTH LOCUST,66762
231-0850
40 M 33002 64 U
ZABEL, KENNETH P, 909 CENTENNIAL,66762
231-1650
37 M 1902 65 IM

PLAINVILLE—913
(Central Kansas Society)

PAGE, O VALE, 409 S COCHRAN ST,67663
434-4609
20 M 1902 51 FP
PEDERSON, ARNOLD M, 409 COCHRANE,67663
434-4609
22 M 1902 51 FP

PLEASANTON—913
(Bourbon County Society)

JUSTUS, WILLIAM J, ,66075
352-6134
29 M 1902 55 FP

PRATT—316
(Pratt-Kingman Society)

AMBLER, CARL D, 200 COMMODORE,67124
-
31 M 1902 57 R
BARKER, PATRICK N, 420 COUNTRY CLUB RD,67124
672-7411
45 M 1902 71 GS
BLACK, CYRIL V, 223 E 4TH,67124
672-6403
05 M 4802 30 GS
FILLEY, VERNON W, 310 E 2ND,67124
672-5555
13 M 3005 43 GS
FREEMAN, F GILES, 310 E 2ND,67124
672-5555
18 M 1902 44 FP
JACKS, J WARREN, 602 E 2ND,67124
672-5559
23 M 1902 48 FP
PITMAN, WILL D, 717 WEST 3RD,67124
-
98 M 1902 25 OO
QUENZER, RONALD W, 420 COUNTRY CLUB RD,67124
672-7411
46 M 1601 73 IM
SIBALA, JUSTO L, 200 COMMODORE,67124
672-7159
20 M 74802 49 R
SUITER, DANIEL JAY, 420 COUNTRY CLUB,67124
672-7411
44 M 1902 71 GE
THOMAS, R CULLEN, 420 COUNTRY CLUB RD,67124
672-7411
47 M 1902 72 GS
THORPE, FRANCIS A, 310 E 2ND,67124
672-5555
08 M 1606 35 FP
WOLFF, FREDERICK P, 223 E 4TH,67124
672-6403
20 M 1902 44 IM

PROTECTION—316
(Iroquois County Society)

GLENN, LYLE G, 145 BROADWAY BOX 447,67127
622-4686
12 M 1606 40 FP

QUINTER—913
(Northwest Kansas Society)

GUNTER, CARL C, QUINTER CLINIC BLDG,67752
754-3333
20 M 1902 51 FP
HIESTERMAN, HERMAN W, QUINTER CLINIC BLDG,67752
754-3333
23 M 1902 51 FP

RANSOM—913
(Central Kansas Society)

MCLAIN, KENNETH, BOX 247,67572
731-2295
21 M 1902 46 FP

RILEY—913
(Riley County Society)

OLTMAN, THEODORE V, RR 1 BOX 145B,66531
485-2549
00 M 1601 29 FP

RUSSELL—913
(Central Kansas Society)

MERKEL, EARL D, SHIELDS BLDG,67665
483-2178
32 M 1902 57 FP
PANICHABHONGSE, SAMBHUNDH, 213 WEST 7TH,67665
483-2178
43 M 89101 67 GS
PETTIJOHN, WALTER J, 624 W 12TH,67665
-
12 M 1902 37 OO
STARKEY, JERALD L, SHIELDS BUILDING,67665
483-2178
30 M 1902 56 FP
SWANN, CLAIR L, 112 W 7TH,67665
483-4212
13 M 1902 39 IM
WHITE, FAGAN N, 356 W 5TH,67665
-
11 M 702 36 OO

SABETHA—913
(Northeast Kansas Society)

BROWN, VIRGIL E, 101B MAIN ST,66534
284-2141
06 M 1902 37 FP
MONTGOMERY, THOMAS ALLEN, 101B MAIN ST,66534
284-2141
10 M 1902 49 FP

SALINA—913
(Saline County Society)

ALLEN, MONTE L, 600 S SANTA FE,67401
827-0307
36 M 1902 61 OTO

ANDERSON, JOOY, 737 E CRAWFORD,67401
827-7261
32 F 1902 59 1M
BAXTER, W REESE, P O BOX 1707,67401
825-8221
47 M 1902 73 FP
BROWN, ROBERT WAYNE, PO BOX 1747,67401
827-5591
23 M 1902 55 1M
BRUMMETT, RICHARD R, P O BOX 1707,67401
825-8221
34 M 1902 64 FP
BRUNGARDT, BERNARD A, 400 E BELOIT,67401
827-4433
21 M 3005 46 ANES
CATHCART-RAKE, WILLIAM F, 737 E CRAWFORD,67401
827-7261
48 M 1902 74 1M
CLARK, DAVID H, 617 E ELM,67401
825-8221
36 M 1902 62 FP
COFFEY, ROY B, 671 ELMORE DRIVE,67401
823-6397
24 M 1902 47 ORS
CONNELLY, MAURICE R, 539 S SANTA FE,67401
827-0268
12 M 2002 38 GS
CONNOR, BRIAN, 1518 B EAST IRON,67401
825-2272
M OPH
COVERT, THOMAS J, PO BOX 360,67401
827-7261
45 M 1902 71 PD
CULTRON, FRANK T, 900 E CRAWFORD,67401
823-8151
10 M 1643 38 OPH
D'SOUZA, BISMARCK C, 116-A S 7TH,67401
827-9526
45 M 49501 67 R
DOWELL, JAMES C, 645 E IRON,67401
827-7255
26 M 1611 49 1M
DRAEMEL, H RICHARD, 600 S SANTA FE ST,67401
827-0307
18 M 1902 53 OTO
DREHER, HENRY S, 737 E CRAWFORD,67401
827-7261
18 M 1902 43 1M
EATON, GLEN E, RFD 6,67401
827-3064
28 M 1902 54 ANES
EATON, LESLIE F, RR 1 BOX 75,67401
827-7261
06 M 1902 32 GS
ELLISON, PAUL D, 1499 E IRON,67401
825-7271
35 M 2105 60 OPH
FORSTER JR, LOUIS G, P O BOX 1707,67401
825-8221
47 M 1902 73 FP
FREEMAN, RAYMOND S, 737 E CRAWFORD,67401
827-7261
20 M 702 50 PD
GANS, FREDERICK A, 737 E CRAWFORD,67401
227-7261
22 M 2834 46 PD
GRIFFING, RICHARD B, 1941C GLENDALE RD,67401
825-9091
27 M 1902 53 ANES
GRIFFITH, FRANK H, 1493 E IRON,67401
827-0488
45 M 4813 75 OPH
GUNN, MARVIN R, 116A S 7TH PO BOX 1059,67401
827-9526
28 M 3901 54 R
GUZMAN, MANUEL, CKMHC,67401
823-6322
27 M 64901 54 P
HARBIN, GARY LYNN, 519 S SANTA FE,67401
827-4424
50 M 1902 75 ORS
HARRIS, NORMAN R, 430 S 7TH,67401
825-8191
30 M 1902 59 OBG
HASSLER, RANDY D, 645 E IRON,67401
827-9635
M U
HATTON, LLOYD W, 617C UNITED BLDG,67401
827-6256
06 M 1902 33 P
HESSE, FREDERICK J, PO BOX #918,67401
827-9631
50 M 1902 75 1M
HOOGES, MERLE A, 430 S 7TH ST,67401
825-8191
34 M 1902 58 OBG
HOLMAN, JON B, PO BOX 61,67401
827-9366
33 M 1902 63 P
JACKSON JR, DELMAS A, 645 E IRON,67401
827-7255
35 M 2101 60 1M
JENKINS, NEAL M, 135 E CLAFLIN,67401
827-9631
19 M 1902 49 1M
KREMBIEL, MARK A, 617 E ELM,67401
825-8221
49 M 1902 74 FP
KRUCKEMYER, ALAN L, 645 E IRON,67401
823-2215
45 M 1103 71 ORS
LAKE, MAX S, 600 S SANTA FE,67401
827-0307
19 M 3005 43 OPH
LASLEY, DAVID A, 645 E IRON,67401
827-9635
22 M 1605 47 U
LIVINGSTON, CHARLES E, PO BOX 918,67401
827-9631
32 M 1611 57 GS
LUNGSTRUM, JACK E, PO BOX 1346,67401
823-2215
21 M 1902 59 ORS
MACY, NORMAN E, RR 3 BOX 37A,67401
827-4053
35 M 1902 60 PATH
MACY, TED L, PO BOX 360,67401
827-7261
43 M 1902 71 GS
MARCHBANKS, DONALD L, 520 COUNTRY CLUB RD,67401
823-2380
24 M 1902 51 FP
MARSHALL, GEORGE W, PO BOX 1705,67401
225-3191
44 M 1902 70 OBG
MARTIN, OLIVER L, 715 E REPUBLIC,67401
827-9631
08 M 1902 37 OBG
MATHIS, JERRY L, 1112 ALBERT,67401
827-9343
35 M 1902 62 PDA
MAXWELL, GORDON E, 135 E CLAFLIN,67401
827-9631
29 M 1902 55 OBG
MCCRAE, SPENCER C, 519 S SANTA FE,67401
827-4424
18 M 3509 43 ORS
MILLER, ELDEN V, 1928 RIDGELEA,67401
827-3061
19 M 1902 44 ANES
MITCHELL, JOHN C, 617 UNITED BLDG,67401
827-3061
13 M 1902 38 GS
MOWERY, WILLIAM E, 737 E CRAWFORD,67401
823-6455
23 M 1902 47 GS
NICKELL, WAITSTILL B, 400 E IRON,67401
827-9618
24 F 1606 50 ANES
NICKELL, WENDELL K, 400 E IRON,67401
827-9618
26 M 1606 50 TS
NULL, WILLIAM G, 135 E CLAFLIN,67401
827-9631
31 M 102 57 PD
OBANDO, GUILLERMO, 2110 KNOLLCREST DR,67401
823-6219
35 M 26404 62 R
PALMER, GERALD K, 1952 RIDGLEA RD,67401
-
24 M 1803 53 PATH
RASSA, REZA P, 204 SEITZ DRIVE,67401
827-4411
33 M 51701 59 R
REECE, RICHARD J, 116A S 7TH,67401
827-9526
23 M 1902 49 R
RODERICK, JAMES E, 645 E IRON,67401
827-9635
23 M 1902 47 U
ROMEISER, REX S, 645 E IRON,67401
827-9635
41 M 1902 67 U
RUEB, ANDREW E, 11 CRESTVIEW DR,67401
827-6691
11 M 1606 35 GS

SCHMIOT, RAMON WARNER, 400 E IRON,67401
827-9618
39 M 1902 6S GS
SCOTT, CHESTER E, S19 S SANTA FE,67401
827-5549
23 M 1902 S1 FP
SEBREE, STEVEN G, PO BOX 360,67401
827-7261
47 M 1902 73 OBG
SIMPSON, J COLBERT, 308 WEST SOUTH,67401
-
07 M 3006 38 OO
SLOO, MILO G, PO BOX 1346,67401
823-221S
41 M 1902 67 ORS
SMITH, BOYO E, BOX 1285,67401
827-40S3
46 M 300S 72 PATH
SMITH, HAROLO R, PO BOX 360,67401
827-7261
19 M 1902 S1 GS
TAYLOR, THOMAS F, 430 S OHIO,67401
827-0346
26 M 1902 53 FP
WAGENBLAST, HOWARD R, 737 E CRAWFORO,67401
827-7261
2I M 1902 49 FP
WATERS, CLARENCE N, S30 S STH,67401
823-6497
13 M 2834 48 O
WEBER, CLARENCE J, 1S6 OVERHILL RO,67401
-
00 M 1902 39 OO
WEBER, ROBERT W, 64S E 1RON,6740I
827-72SS
26 M 1902 49 IM
WEBER II, RALPH H, 737 E CRAWFORO,67401
827-7261
44 M 300S 7S PO

SATANTA—316
(Southwest Kansas Society)

JABEL, JUVENAL T, SATANTA CLINIC,67870
649-2771
43 IM
TAOURAN, VIRGILIO, SATANTA CLINIC,67870
649-2771
43 M 74810 69 PATH

SCOTT CITY—316
(Southwest Kansas Society)

ARROYO, ZEFERINO, SCOTT CITY CLINIC,67871
872-2187
M 74802 GS
OUNN, OANIEL R, 202 COLLEGE,67871
462-3332
49 M 1902 26 FP
FIELOS, GALEN W, 1106 COLLEGE,67871
872-2187
1S M 1902 49 FP
HOPKINS JR, B MORRISON, 804 CRESCENT,67871
872-2187
23 M 1902 S3 FP

SEDAN—316
(Southeast Kansas Society)

LIM, CARLO, 104 W MAIN,67361
725-3171
44 M 74801 70 IM
TAYLOR, ELMER W, 120 WEST OSAGE,67361
72S-3I41
28 M S12 57 FP
WALKER, WILLIAM K, 111 E CHEROKEE,67361
725-3171
I8 M 1902 4S FP

SENECA—913
(Northeast Kansas Society)

BERKLEY, NORMAN W, 1S SOUTH STH ST,6653B
336-2128
31 M 1902 63 FP
GILBERT, J HOWARD, 603 N FIFTH,66S3B
336-24I4
0S M 1902 41 FP
LUEGER OO, JAMES JOHN, 201 N SIXTH,66S3B
336-6113
S1 M 2878 78 GP

SHARON SPRINGS—913
(Northwest Kansas Society)

CHUNG, JOHN J, WALLACE CO MED CLINIC,677SB
852-4214
23 M SB301 48 FP

SHAWNEE MISSION—913
(Johnson County Society)

ALLEN JR, LEWIS G, 7301 MISSION RD SUITE 300,66208
362-2222
21 M 1902 45 R
ALLEN JR, WILLIAM R, 6734 GRANADA RO,66208
-
46 M 1902
ALTENBERNO, ELVIN CONRAO, 7319 W BIST,66204
648-20I0
26 M I902 S4 FP
ALVAREZ, LUIS A, 8100 MARTY,66204
649-3400
32 M 64914 6I FP
ARMBRUSTER, ALBERT A, 9119 WEST 74TH SUITE 202,66204
362-9220
17 M S12 55 GS
ATHON, MERRILL O, 8501 OELMAR,66207
642-4242
24 M 1902 54 FP
BAOEEN II, LOUIS JOHN, 10SS0 QUIVIRA RO,6621S
492-3344
49 M 2820 74 OPH
BAEKE, JOHN O, 6806 WEST B3RO,66204
642-4242
19 M 1902 S2 FP
BAKER, WILLIAM STEVEN, 7700 W 63RO,66202
SB8-6489
47 M 702 73 P
BALANOFF, ARNOLO Z, 4601 W 109TH SUITE 122,66211
642-4040
42 M 1803 67 PO
BANSAL, SATISH C, 6100 MARTWAY,66202
384-2220
3B M 49S41 61 ORS
BAPTIST, JEREMY E, S811 OUTLOOK,66202
432-062S
40 M 2846 78 A
BARE II, CHARLES E, 8901 W 74TH SUITE 3S3,66204
764-3081
43 M 1902 69 U
BARKER, ELIZABETH B, 4121 WEST B3RD SUITE 123,66208
381-6669
30 F 4706 S5 P
BARKER, JAMES BERTON, 8901 W 74TH,66204
362-6310
31 M 4706 SS OTO
BARNHART, RONALO J, 9119 WEST 74TH,66204
831-2334
41 M 2S0I 68 OBG
BARR, RICHARD N, 7301 MISSION ROAO,66208
432-4366
32 M 1902 57 OPH
BARRICK, BRUCE, SH MISSION MEDICAL CENTER,66201
676-2340
39 M 1902 65 PATH
BATTY, THOMAS V, SSSS W SBTH,66202
432-2080
21 M 3806 S4 FP
BEAMON, RICHARO F, 10500 QUIVIRA,66215
492-1000
47 M 2803 73 EM
BELL, OELORIS W, 4601 WEST 109TH SUITE 104,66211
341-6SS0
42 F 1902 68 OPH
BELZER, EOWARD G, 4601 WEST 109TH SUITE 110,66211
381-8282
36 M 300S 58 PO
BIKALES, VICTOR WILLIAM, 7301 MISSION RO,6620B
384-1311
13 M 2105 38 P
BISHOP, FRANCIS E, 7S01 MISSION ROAO,6620B
648-3S33
20 M 1902 4S P
BLETZ, OONALO B, 105S0 QUIVIRA RO/STH FL,65215
492-6200
28 M S104 SB IM
BOLES, J MICHAEL, 5949 NIEMAN ROAO,66203
631-1300
3S M I902 61 FP
BRAVERMAN, OAVIO ELLIOTT, 4601 W 109,6621I
341-1101
47 M 2S07 72 POO
BRIOGENS, JAMES G, SH MISSION MEDICAL CENTER,66201
676-2340
22 M 1902 47 PATH

BROWN, WILLIAM R, 7301 MISSION RD,66208
236-8866
23 M 1902 48 IM
BROXTERMAN, STEVEN JOSEPH, 8901 W 74TH,66204
362-5510
51 M 1902 76 FP
BRUNING, ROGER MARION, 7301 MISSION SUITE 342,66208
384-0745
48 M 1902 70 FP
BUCKMAN, MARTIN SPALDING, 10550 QUIVIRA SUITE 290,66215
492-6200
49 M 2802 76 IM
BURGER, PAUL B, 5638 NIEMAN ROAD,66203
631-6114
25 M 2834 50 FP
BURKET JR, GEORGE E, 2012 CONDOLEA DR,66209
-
12 M 1902 37 OD
BURTON, JERALD ALBERT, 10550 QUIVIRA,66215
492-6200
42 M 2845 73 IM
CAIN, IVAN W, 4200 SOMERSET STE 224,66208
649-4599
18 M 1902 44 ORS
CALKINS, LARRY L, JOHNSON CO NATL BK BLDG,66207
649-0110
18 M 1902 43 OPH
CARDUFF, JAY J, 6300 GLENWOOD,66202
432-4480
25 M 3006 54 FP
CAREY, LARRY J, 2624 WEST 77TH,66208
-
51 M 1902 *
CARRASCO, LENOR C, 8971 W 75TH, 66204
341-2000
41 F 74801 63 PD
CAUGHRON, MICHAEL ROBERT, 5105 NEDSHO,66205
-
49 M 1902 74 PATH
CAVITT, ROBERT F, 9119 WEST 74TH,66204
831-0700
24 M 1902 48 GS
CEDERLIND, CRANSTON JAY, 8901 W 74TH #248,66204
384-4990
45 M 1902 71 OBG
CHANG, SHU FANG, 10200 W 75TH,66204
432-7885
25 F 24239 49 P
CHRISTIAN, STANLEY J, 9129 DELMAR,66207
371-1181
18 M 1902 44 FP
CLENDENIN, ROBERT KEELE, 4707 W 110TH,66211
648-6060
48 M 1902 73 EM
COE, RICHARD O, 7301 MISSION RD SUITE 247,66208
362-8505
31 M 4804 56 OPH
COHEN, ROBERT A, 3700 W 83RD,66208
642-2100
39 M 2803 64 PD
COHN, STEVEN G, 8627 LINDEN DR,66207
334-2500
41 M 1902 67 ANES
COOPER, JACK R, 7301 MISSION RD SUITE 330,66208
432-6300
17 M 3840 43 NS
COOPER, LEO F, 3819 W 57TH TERR,66205
588-6766
15 M 1902 53 FP
CORBIN, MURRAY D, 10550 QUIVIRA RD-5TH FL,66215
492-6200
39 M 1902 65 IM
COULTER, HENRY F, 5829 WOODSON PO BOX 975,66201
722-1100
23 M 1902 51 FP
COX JR, IRA, 5829 WOODSON PO BOX 975,66201
722-1100
19 M 1902 49 FP
CURRAN, KEVIN E, 4121 W 83RD,66208
649-9383
39 M 2803 65 OPH
DAVIS, RICHARD E, 10955 GRANADA LN,66211
648-5303
26 M 1902 54 P
DELP, MAHLON H, 6131 TERRYDALE RD,66202
-
03 M 1902 34 IM
DENISON, TERRY R, 5811 OUTLOOK,66202
432-0625
29 M 1902 56 PD
DERRINGTON, KENNETH L, FOX HILL MED BLDG,66211
341-3535
44 M 1902 71 FP
DOCKHORN, ROBERT J, 5300 W 94TH TERR,66207
381-4674
34 M 1902 60 POA
DOHERTY, WILLIAM R, 7600 STATE LINE,66208
649-3900
20 M 3006 56 FP
DUCKETT II, THOMAS G, 4601 W 109TH SUITE 116,66211
648-1022
41 M 1902 67 OPH
EIKERMANN, WILLIAM C, 9400 MISSION RD,66206
642-5184
42 M 1902 69 P
ENDERS, WRAY, 9034 COTTONWOOD DR,66215
-
02 M 1902 36 ANES
ESRIG, HAROLD L, O.D., 8132 SAGAMORE,66206
381-5033
30 M 2878 60 ANES
ETZENHOUSER III, RUSSELL D, P O BOX 7426,66207
381-8282
34 M 1902 59 PD
EVANS JR, WILLIAM E, 8741 HIGH DRIVE,66206
362-7363
24 M 1902 58 FP
FELT, SAMUEL E, 2700 W 120 TERRACE, 66209
-
46 M 1902 72 00
FERGUSON, ROBERT LEON, 8901 W 74TH SUITE 208,66204
362-6161
47 M 1902 73 IM
FORDYCE, NORMAN, 8901 W 74TH,66204
722-0020
41 M 1902 67 DTD
FRANCISCO, CLARENCE L, 3509 W 85TH,66206
371-6802
09 M 1902 34 ORS
FULLEN, JERYL G, PO BOX 4010,66204
831-3500
43 M 401 68 DRS
GALLEHUGH, KEITH W, 9027 BIRCH,66207
371-4343
32 M 1902 57 R
GARCIA, FRANCISCO, 8020 SANTA FE,66204
642-5000
32 M 27501 60 FP
GARDNER, GLENN M, 5200 WEST 64TH,66208
-
35 M 2803 60 IM
GENTRY, KALE C, 5105 W 84TH,66207
632-4242
31 M 1902 60 FP
GILBERT, ROBERTA M, GEORGETOWN MEDICAL BLDG,66204
341-1234
35 F 3505 62 P
GILLEN, BILLY A, 8802 BIRCH LANE,66207
-
29 M 1902 54 ANES
GOLLERKERI, MOHAN P, 7301 MISSION RD STE 339,66208
236-8866
30 M 49516 52 HEM
GOMEZ, FRANCISCO, 4200 SOMERSET #160,66208
649-7300
15 M 26401 43 P
GOOD, WENDELL LISLE, 4601 W 109TH,66211
649-3883
24 M 1902 48 FP
HAAS, JUDITH S, 5200 W 67TH,66208
588-7350
45 F 1902 71 RO
HACKER, DAVID CHARLES, 5916 MISSION RD,66205
588-6670
50 M 1902 75 ANES
HAMTIL, LAWRENCE W, 10550 QUIVIRA RD,66215
649-6722
36 M 2803 61 PD
HANDLEY, DENNIS MICHAEL, 8650 W COLLEGE, 66215
649-1311
50 M 2803 76 FP
HARMS, ALBERT C, 5750 WEST 95TH,66207
381-5550
13 M 1902 38 FP
HARPSTER, GENE O, 4601 W 109TH,66211
648-1400
31 M 1902 57 GS
HARTONG, WILLIAM ALLEN, 9119 W 74TH,66204
831-9300
44 M 1902 71 IM
HASTINGS, MARY T, 2520 W 50TH PL,66205
-
45 F 1902 77 D

HATHAWAY, PETER, 11055 CEDAR STE 216,66211
383-2270
31 M 3503 60 IM
HENRY, JOSEPH E, 8901 W 74TH,66204
432-8000
42 M 1902 68 PUO
HERZON, CHARLES O, 8951 W 75TH,66204
649-9010
39 M 3005 65 OBG
HESSER, HERBERT H, 7207 EDGEWOOD BLVD,66203
-
06 M 1902 34 OO
HEIBERT, PETER E, 9021 SALEM DR, 66215
371-4343
03 M 1902 34 R
HOBSON, MILBURN W, 9119 W 74TH ST,66204
831-2334
30 M 1902 55 OBG
HODES, HERBERT C, 5007 W 112TH,66211
381-6868
43 M 1902 69 OBG
HODGES, BRUCE E, PO BOX 5355,66215
888-0777
32 M 1902 63 FP
HOPKINS, WILLIAM O, 10000 W 75TH,66204
831-3500
33 M 2803 61 ORS
HOPKINS JR, LENLY T, 8600 W 95TH ST,66212
649-7844
30 M 3841 56 GS
HORSEMAN, ROBERT F, 9119 W 74TH ST,66204
432-7419
19 M 1902 44 OBG
HOUSTON II, LAWRENCE MORLEY, 8650 W COLLEGE 66215
649-1311
50 M 2803 76 FP
HUMPHREY, LOREN JENKINS, 9119 W 74TH,66204
531-2514
31 M 1611 56 SON
HYLWA, THEODORE M, 10460 MASTIN,66212
492-3266
43 M 1902 69 PS
ITURRALDE, GEORGE, 7501 MISSION RD,66208
648-4949
21 M 13201 49 P
JANES, DONALD R, 10550 QUIVIRA #360,66211
492-1955
34 M 1902 60 OBG
JOHNSON, NADINE KAY, 10550 QUIVIRA,66215
492-6200
38 F 1803 63 IM
JONES, CHARLES E, SHAWNEE MISSION MED CNTR,66201
676-2214
31 M 1902 60 FP
JONES, H IVOR, 8901 W 74TH SUITE 269,66204
362-4040
24 M 80303 51 P
JOUVENAT, NEIL C, 10550 QUIVIRA SUITE 120,66215
492-1844
43 M 3005 71 OBG
KADIAN, RAJESH S, 10550 QUIVIRA 5TH FLOOR,66215
492-6200
50 M 71 IM
KAGAN, STUART M, 10550 QUIVIRA - SUITE 340,66215
492-1111
44 M 4901 69 PD
KASHYAP, BANSHI PRASAD, 8901 W 74TH, 257, 66204
236-4500
47 M 49554 69 IM
KENNEDY, KENNETH R, 6100 MARTWAY STE 11,66202
432-0126
24 M 1902 53 FP
KETCHUM, LYNN O, 10550 QUIVIRA RD STE 310,66215
492-3737
36 M 2101 60 PS
KHURANA, SATISH K, 9119 W 74TH,66204
432-3334
41 M 49536 65 PD
KIMURA, CHARLES C, 8901 W 74TH,66204
262-4220
25 M 2101 56 A
KODANAZ, A AYTEKIN, 5710 REINHARDT DR,66205
334-2500
28 M 90201 55 ANES
KOZIKOWSKI, BEN M, 7301 MISSION RD,66208
362-8317
30 M 2834 55 ORS
KRUEGER, KURT ALLEN, SHAWNEE MISSION MED CNTR,66201
676-2479
48 M 3006 74 ANES
KUBIN, DORIS A, 2504 W 71ST,66208
371-4343
15 F 1902 43 R

KURTH, ROBERT H, 5555 W 58TH,66202
432-2080
28 M 3005 53 IM
LAPI, RUTH M, 2012 STRATFORD RD,66208
-
14 F 4107 37 OO
LEATHERS III, HOLLIS K, SHAWNEE MISSION MED CNTR,66201
676-2340
38 M 3901 64 PATH
LEGASPI JR, PEDRO L, SHAWNEE MISSION MED CNTR,66201
676-2479
36 M 74801 60 ANES
LEIGH, LAWRENCE E, 8020 SANTA FE DR,66204
642-4080
12 M 1902 41 FP
LESTER, JOHN BUCKLES, 4140 W 71ST SUITE 108,66208
432-7276
45 M 1902 70 P
LEWIN, WALTER, 8901 W 74TH ST,66204
362-4040
30 M 1902 56 P
LEWIS, JAMES E, 3700 W 83RD SUITE 203,66208
649-0923
37 M 2101 63 P
LIPSEY, JAMES H, 10000 W 75TH SUITE 103,66204
831-3500
31 M 1606 56 ORS
LOREN, MICHAEL LEWIS, 5300 W 94TH ST TERR,66207
381-4674
48 M 2507 73 PED
LULO, ANTONIO R, 7600 STATE LN,66208
649-3900
35 M 30801 60 IM
MALLORY, JOHN A, 10550 QUIVIRA 5TH FLOOR,66215
492-6200
43 M 2803 71 IM
MANLEY, JOSEPH W, 4601 W 109TH SUITE 306,66211
381-8838
42 M 1902 69 OBG
MANTZ, FRANK A, 9309 W 103RD,66212
588-7099
12 M 4101 38 OO
MASER, GEORGE R, 5811 NALL,66202
432-5515
12 M 1902 36 FP
MATHEWS, ROBERT MAJOR, 7301 MISSION RD,66208
362-6888
25 M 1902 54 GS
MAXWELL, ROBERT A, 8901 W 74TH - SUITE 150,66204
362-1660
46 M 1902 73 PD
MCCAUGHEY, HUGH W, 11055 CEDAR SUITE 210,66211
381-1724
28 M 1902 53 IM
MCEACHEN, WILLIAM H, 3700 WEST 83RD SUITE 102,66208
649-3335
32 M 1902 59 PD
MCELROY, MICHAEL B, 6300 GLENWOOD,66202
432-1122
43 M 2803 69 ORS
MCGURK, THOMAS E, 4601 W 109 SUITE 206,66211
649-2080
39 M 2803 65 P
MCWHERTER, LOTTIE B, 5920 NALL SUITE 308,66202
362-1464
30 F 1902 57 IE
MELIA JR, B JAMES, 3700 W 83RD,66208
648-4117
33 M 2803 60 O
MORANTZ, ROBERT A, 8420 ROE, 66207
588-6119
42 M 3519 67 NS
MENEZ, CESAR V, 4121 W 83RD SUITE 120,66208
381-4484
36 M 74810 56 P
MILLER, FREEMAN LANCE, DOCTOR'S BLDG,66215
492-6200
48 M 1902 74 PD
MORONEY, JEAN M, 10550 QUIVIRA,66215
492-6200
25 F 4109 65 N
MUEHLBERGER, JAMES J, 4601 W 109TH SUITE 314,66211
383-3222
34 M 3006 60 PD
MUELLER, J KENT, 3700 WEST 83RD SUITE 203,66208
649-0923
35 M 1902 62 P
NASH, ROBERT A, 4601 W 109TH,66211
649-8686
31 M 1902 55 P
NAUER, PAULA LOU, 7301 MISSION RD STE 342,66208
384-0745
49 F 1902 74 FP

NEIBURGER, JAMES B, 5300 W 94TH TERR,66207
381-4674
46 M 1642 72 A
NELSON, BRYAN C, 9119 W 74TH,66204
384-5500
50 M 1902 75 PD
NIEMAN, JOHN L, PO BOX 7426,66207
381-8282
28 M 3805 58 PD
NOSTI, JUAN C, 8901 W 74TH #345,66204
262-5014
38 M 13204 63 PS
NYE, C ERIK, 7301 MISSION RD SUITE 348,66208
362-8317
39 M 3520 65 ORS
O'BRYAN, JAMES J, 10550 QUIVIRA,66215
492-2525
47 M 1902 73 PD
O'CONNELL, FRANK A, 7830 STATE LINE,66208
381-0886
25 M 1902 51 FP
OKTAWIEC, DANUTA, 5848 FONTANA DRIVE,66205
22 F 80303 50 ANES
OSGOOD, GEORGE M, 3700 WEST 83RD,66208
381-5200
14 M 1902 44 IM
PATTERSON, JOHN R, 5317 CHADWICK RD,66205
20 M 1902 48 PD
PEARCE, EUGENE W J, 9119 W 74TH SUITE 104,66204
722-3102
24 M 2802 49 OBG
PEARCE, LUNETTA M, 9119 W 74TH,66204
362-1525
26 F 3005 49 FP
PENTECOST, RICHARD L, 8900 STATE LINE SUITE 350,66206
383-1410
32 M 1001 56 P
PERICO, CARLOS J, 9209 ROE, 66207
888-0777
32 M 26404 60 FP
PETERSEN, A GENE, 3700 W 83RD,66208
648-3911
27 M 1902 54 IM
PETERSEN, GERALD D, 3700 WEST 83RD,66208
648-3911
30 M 1902 60 IM
PETIT, CARL ALFONSO, 10550 QUIVIRA,66215
492-6200
33 M 60 GS
PETRIE, SAMUEL C, GEORGETOWN MED BLDG,66202
722-1166
27 M 1902 58 IM
PETTEGREW, PAULINE K, 5600 W 95TH SUITE 105,66211
381-6765
21 F 3006 50 FP
PFUETZE, BRUCE L, 4601 W 109TH,66211
383-3630
42 M 1902 68 A
PFUETZE, KARL O, 10550 QUIVIRA 5TH FLOOR,66215
492-6200
40 M 1902 66 CD
PHILLIPS, WARREN G, 3700 W 83RD,66208
649-0923
26 M 1902 60 P
PILCHARD, WILLIAM A, GEORGETOWN MED BLDG,66204
632-3210
39 M 1602 65 OPH
PITTS, RONALD L, 8901 W 74TH - SUITE 330,66204
362-2524
35 M 2002 62 O
POWELL, CAROL W, 8216 CHEROKEE CIRCLE,66206
381-3785
25 F 1902 51 P
POWELL, KENNETH A, 8216 CHEROKEE CIRCLE,66206
753-7000
25 M 1902 53 IM
PRONKO, MICHAEL J, 4121 W 83RD SUITE 223,66208
648-7878
34 M 1902 60 P
RACELA JR, ANTONIO S, SUBURBAN MEDICAL CENTER,66215
492-1000
37 M 74802 63 PATH
RADOM, SANFORD B, 2304 W 121ST,66209
677-0883
40 M 1642 66 R
REIVICH, RONALD S, 11030 GRANADA LANE,66211
383-3050
34 M 3806 60 P
RIEKE, FRANK A, 10411 WEST 55TH,66203
16 M 1902 41 GS

ROSENBERG, STANTON L, 1900 W 75TH SUITE 200,66208
362-8080
30 M 1902 55 P
ROSENTHAL, RICHARD, 5102 W 112TH ST,66211
492-1000
50 M 2820 76 EM
RUBIN, HERBERT M, 10550 QUIVIRA - SUITE 340,66215
492-1111
37 M 2802 63 PD
RUMOLD, MERVIN J, 6340 INDIAN LANE,66208
03 M 1902 30 GS
SAFFD, KARL S, 8901 W 74TH,66204
362-9585
39 M 52801 62 PS
SATHYANARAYANA, SARASWATHI, 9119 W 74TH,66204
432-7419
45 F 49509 67 OBG
SAWKAR, LAXMIDAS A, 10314 CATALINA,66207
384-4844
36 M 49523 63 IM
SCHAEFER, JOSEPH PETER, 10550 QUIVIRA - SUITE 230,66215
492-7440
34 M 1902 60 IM
SCLAR, WILLIAM C, 7301 MISSION RD SUITE 225,66208
362-7900
46 M 2501 72 GS
SHAAD, DOROTHY J, 2322 W 51ST,66205
09 F 1902 44 OO
SHOFSTALL, WILLIAM H, 6100 MARTWAY,66202
722-4747
11 M 3901 41 ENT
SMITH, DALE C, 4601 W 109TH ST SUITE 224,66211
381-0353
20 M 1902 45 OPH
SMITH, DONALD J, 8600 W 95TH ST,66212
642-4515
18 M 1902 49 FP
SNODELL, FIRMIN E, 5555 W 58TH,66202
432-2080
31 M 1902 61 IM
SNOW JR, ARTHUR D, 8901 W 74TH - SUITE 225,66204
362-5510
45 M 1902 75 FP
SOELDNER, JAMES OLIVER, 9124 DEARBORN, 66207
44 M 1902 70 FP
SPEER, FREDERIC, 5811 OUTLOOK,66202
432-0625
09 M 1902 34 A
STEINER, ROBERT M, 5311 W 96TH TERRACE,66207
677-0883
39 M 4112 65 R
STEINZEIG, ALFRED S, 6500 HODGES DR,66208
13 M 1902 37 OO
STEVENSON, E KENT, 4121 W 83RD SUITE 150,66208
649-5566
45 M 2802 67 CHP
STITT, RONALD W, 4221 W 104TH TERR, 66207
342-7233
21 M 1902 45 U
STUBER, JACK LAWRENCE, PO BOX 4250,66204
677-0883
40 M 1902 66 OR
SUGAR, ROBERT L, 8901 W 74TH #248,66204
384-4990
40 M 3508 66 OBG
SULLIVAN JR, HENRY B, 5817 NIEMAN RD,66203
631-6160
24 M 1902 52 FP
SUTTON JR, RICHARD L, 3203 W 83RD TERR,66206
08 M 2501 29 OO
TETZLAFF, ARCHIBALD D A, 4520 W 65TH,66208
334-2500
26 M 40721 52 ANES
THOMSEN, GARY, 8901 W 74TH,66204
362-5510
51 M 3005 76 FP
TRETBAR, LAWRENCE L, 8901 W 74TH,66204
677-1776
33 M 1902 60 GS
TROPP OD, ARNOLD L, 8600 W 95TH STE 102,66212
648-5600
46 M 2878 72 FP
TUTERA, GINO, 10550 QUIVIRA SUITE 120,66215
492-1844
45 M 2803 71 OBG
VALK, WILLIAM L, 5401 W 81ST,66208
588-6146
09 M 2501 37 U

VANNAMAN, DONALD D, P O BOX 7426,66207
381-8282
43 M 1902 71 PD
VELARDE, HUGO, 6100 MARTWAY,66202
287-8400
M 64 GS
WALKER, WILLIAM L, 10500 QUIVIRA,66209
492-1000
44 M 1902 70 R
WANG, SIDNEY W, 9119 W 74TH ST SUITE 102,66204
722-2020
32 M 38503 58 FP
WEARE, MARY E, 7301 MISSION RD #304,66208
362-7224
48 F 4002 73 P
WEBB, JAMES R, 5949 NIEMAN ROAD,66203
631-0900
34 M 1902 61 FP
WERTH, CLAUDE J, 4121 W 83RD,66208
649-5566
37 M 1902 64 P
WHITEHEAD, RICHARD E, 7301 MISSION RD,66208
362-8317
31 M 2501 58 ORS
WHITLEY, DOUGLAS M, 4601 W 109TH SUITE 202,66211
341-4770
34 M 1902 60 D
WIGGINTON, GERALD D, D.O., 9119 W 74TH,66204
384-5500
44 M 2878 70 PD
WILEY, JOHN H, 9119 W 74TH ST,66204
831-2334
37 M 4113 63 OBG
WILSON, L BARRICK, 5602 FAIRWAY RD,66205
236-8423
09 M 1902 35 ANES
WILSON, ROBERT B, 7301 MISSION,66208
362-3356
10 M 1902 40 ENT
WILSON, SLOAN J, 5618 W 62ND,66202
-
10 M 1902 36 HEM
WURSTER, GEORGE R, 3700 W 83RD SUITE 203,66208
649-0923
35 M 1902 61 P
YE, RICHARD C, 7301 MISSION RD,66208
362-7505
20 M 24222 46 PS
YOHE, RUTH M, 8600 W 95TH ST,66212
383-3377
26 F 4107 54 PDA
YOUNG, JOHN W, 7301 MISSION RD SUITE 317,66208
362-7505
37 M 4706 63 PS
YOUNGLOVE, R HAL, 10550 QUIVIRA SUITE 120,66215
492-1844
50 M 3005 75 OBG
YOUNGSTROM, KARL A, 9660 REEDER PLACE,66214
-
08 M 3607 44 DO
ZACK, ASHLEY S, 4601 WEST 109TH SUITE 122,66211
642-4040
46 M 2803 73 PD
ZAMIERDWSKI, DAVID S, 10550 QUIVIRA,66215
362-7290
42 M 2307 68 PS
ZAREMSKI, SHERMAN C, 7301 MISSION RD SUITE 214,66208
362-8566
33 M 1720 58 IM
ZIMMERMAN, DANIEL D, 1900 W 47TH PL SUITE 310,66205
236-7810
45 M 3005 70 IM

SMITH CENTER—913
(Central Kansas Society)

RELIHAN, FRANCIS H, 116 SOUTH MAIN,66967
282-3291
84 M 1606 07 FP
SHEPPARD, ROBERT G, 120 E COURT,66967
282-6654
21 M 1902 45 GS
STEINKRUGER, VERLYN WILLIAM, 120 E COURT,66967
282-6654
28 M 3005 53 FP
WOODS, HUGH J, 120 EAST COURT,66967
282-6654
26 M 1902 52 FP

SOUTH HAVEN—316
(Tri-County Society)

UBELAKER, ERNEST J, ,67140
892-2261
11 M 1902 38 FP

ST. FRANCIS—913
(Northwest Kansas Society)

CRAM, ERNEST R, PO BOX 625,67756
332-2126
24 M 1902 52 FP
JEWELL, ROSS L, 203 SPENCER,67756
332-2832
13 M 1902 56 FP
STEPHENSON, LUCILLE C, ,67756
-
06 F 1902 32 FP
WALZ, THOMAS J, 115 SOUTH QUINCY,67756
-
94 M 1902 21 OD

ST. JOHN—316
(Stafford County Society)

QUACKENBUSH, ROBERT P, ,67578
549-3471
21 M 1611 52 FP
SCHERER, ALFRED L, 610 E FIRST,67576
549-3261
32 M 1902 57 FP

ST. MARYS—913
(Pottawatomie County Society)

BROWN, FRED E, 602 W PALMER,66536
437-2256
26 M 1902 55 FP

STAFFORD—316
(Stafford County Society)

BROWN, C EVERETT, 102 N MAIN,67578
234-5251
10 M 1902 47 A
KAYLOR, MARIETTA J, 602 S BUCKEYE,67578
234-5805
39 F 0512 66 FP
WARD, ROBERT L, 412 E GRAND,67578
234-5281
24 M 1902 52 FP

STERLING—316
(Rice County Society)

SIMPSON, TOM C, 239 N BROADWAY,67579
278-2123
47 M 1902 73 FP

STOCKTON—913
(Central Kansas Society)

MAUCK, HAROLD C, 623 SOUTH 2ND,67669
425-6280
20 M 1902 54 FP
VOTAPKA, WILLIAM L, 623 S SECOND,67669
425-6280
24 M 1902 53 FP

SUBLETTE—316
(Southwest Kansas Society)

THIEMANN, A H, D.O., 301 DERBY,67877
675-2241
12 M 1805 42 FP

SYRACUSE—316
(Southwest Kansas Society)

PETTERSON, CECIL E, PROFFESIONAL ASSOCIATION,67878
384-5731
14 M 1902 39 FP

TONGANOXIE—913
(Leavenworth County Society)

STEVENS, PHILIP L, BOX 319,66086
845-2090
27 M 1902 54 FP

TOPEKA—913
(Shawnee County Society)

ACKROYD, ALAN W, 918 W TENTH,66604
233-9681
46 M 4901 74 IM
AGAN, LAWRENCE M, 1314 PEMBROKE LN,66604
357-4306
20 M 5002 44 R
ARMSTRONG, A L, 918 W TENTH,66604
233-9681
41 M GS

ARREDDNDD, MARID, 5024 SW 26TH,66614
296-7216
25 M 26401 54 P
ARUNAKUL, PUNYA, TDPEKA MED CENTER,66604
233-9681
44 M 89104 69 DTD
ASHLEY, BYRDN J, 3222 PLASS,66611
233-2280
98 M 1902 24 DPH
ASHLEY JR, B JDHN, 1616 WEST 8TH ST,66606
233-2280
31 M 1902 56 DPH
AVERILL, STUART C, MENNINGER FD,66601
234-9566
24 M 502 52 P
BAEHR, RALPH H, S-F CAP REG RADIDTHER CTR,66606
234-3451
35 M 1606 59 R
BAIR, GLENN D, 815 CDNTINENTAL BLDG,66606
233-5153
31 M 2401 57 IM
BAKER, FREDERICK C, 2101 WEST 10TH,66604
232-0909
35 M 4113 62 FP
BAKER, PHILLIP L, 300 CONT MED BLDG,66606
357-0301
37 M 3005 63 DRS
BAKER, RAY D, TOPEKA-SHAWNEE HEALTH DEP,66606
233-8961
30 M 4812 55 GPM
BARNHILL, C ALTON, MENNINGER FD,66601
234-9566
31 M 3601 56 P
BAUCDM, KARAN YVDNNE, 1700 W SEVENTH,66606
233-1979
50 F 1902 75 DBG
BAUDE, EUGENE L ANDRE, 1815 WESTWDDD CIRCLE,66604
354-1946
02 M 39606 27 IM
BEACH, RICHARD R, 106 MED ARTS BLDG WEST,66604
233-7943
23 M 2802 48 IM
BEALE, DAVID A, MENNINGER FD,66601
234-9566
31 M 5A04 56 P
BEATY, JAMES R, 1700 WEST 7TH,66606
354-1111
32 M 5101 66 EM
BECK, JOSEPH D, 1001 GARFIELD,66604
235-2351
18 M 3005 A3 PD
BEDFDRD, D R, 6423 HUNTDDN,66601
-
09 M 4802 40 DD
BEELMAN, FLDYD C, 1319 HUNTDDN,66604
235-3981
02 M 38A0 35 FP
BELLER, WILLIS L, 310 MED ARTS BLDG WEST,6660A
234-3451
14 M 1902 41 R
BERLAND, DAVID I, BDX 829,66601
234-9566
47 M 2803 78 P
BLAKE, HENRY S, 1933 WESTWDDD DR,66604
-
11 M 3520 37 DD
BDNEBRAKE, CHARLES RICHARD, 1700 W SEVENTH,66606
233-1979
48 M 1606 75 DBG
BDREL, DAVID, ST FRANCIS HDSP & MED CTR,66606
295-8473
A5 M 1902 71 PATH
BDWEN, CLDVIS W, 2200 WEST 10TH,66604
234-8601
12 M 1902 37 FP
BDWEN JR, HARRY J, 2200 WEST 10TH,6660A
234-8601
11 M 1902 37 FP
BDYD, SPENCER H, 1815 WEST 2ND,66606
-
11 M 1902 35 DBG
BRAUNSDDRF, RDBERT L, 2625 DHID,66605
354-7630
08 M 2501 35 FP
BRIDWELL, RUSSELL E, 4715 CEDAR CREST,66606
234-2624
26 M 1902 51 ENT
BRDCHER, TDBIAS, PD BDX 829,66601
234-9566
17 M 40733 42 P
BURDZIK, EBERHARD G, 2700 W SIXTH,66606
296-4222
26 M A0916 5A P

CAPDDTH, L WAYNE, 915 NE 35TH,66617
286-1788
44 M 4706 70 EM
CARVER, LARRY A, 130 CDNT MED BLDG,66606
234-8653
37 M 1902 73 P
CASHMAN JR, MAURICE R, 901 GARFIELD,66606
354-9591
35 M 1902 61 HEM
CAVANAUGH, JOHN W, 200 PROFESSIDNAL BLDG,66604
235-3488
13 M 1803 39 GS
CHAMBERLIN JR, CECIL R, BDX 829,66601
234-9566
30 M 3901 55 CHP
CHEN, TAK-MING, 303 MED ARTS BLDG,66604
234-3451
41 M 24402 68 ANES
CHERRY JR, ARTHUR C, 918 WEST 10TH,66604
233-9681
27 M 3806 53 PD
CHEUNG, P W H, 918 WEST 10TH,66604
233-9681
36 M 2A338 58 R
CHDY, JAMES K L, 1516 W SIXTH,66606
233-9681
10 M 1643 37 U
CLARK, CRAIG N, 3124 E 6TH,66607
354-7683
29 M 1902 58 FP
CDCHRAN, PAUL W, MENNINGER FD,66601
234-9566
33 M 4802 58 IM
CDHEN, LDUIS, 918 W 10TH,66604
233-9681
14 M 1902 41 IM
CDKELEY, JOHN M, 2200 GAGE BDULEVARD,66622
272-3111
30 M 5501 55 P
CDLLINS, DEAN T, MENNINGER FD,66601
234-9566
28 M 1902 55 P
CDLLINS, EDWARD JDSEPH, 900 WASHBURN,66606
233-3242
45 M 1611 71 DPH
CDLLINS, FRANCIS T, 206 MED ARTS BLDG EAST,6660A
233-6470
14 M 1902 43 IM
CDNNELLY, JOHN C, MENNINGER FD,66601
234-9566
39 M 3806 65 P
CDNRDY, RDBERT W, MENNINGER FD,66601
234-9566
38 M 2604 6A P
CDTTDN, RDBERT T, 901 GARFIELD,66606
354-9591
19 M 1902 45 IM
CRARY, JOHN E, 201 MED PLAZA BLDG,66604
233-A202
18 M 1902 43 IM
CRDUCH, WILLIAM H, 109 MED ARTS BLDG WEST,6660A
232-8224
20 M 2802 45 PD
DAVIS, CHESTER R, 531 HDRNE,66606
232-9394
50 M 1902 75 FP
DE SDIGNE, RAPHAEL R, 2400 W 29TH,66611
267-0150
19 M P
DELGADD, SERGIO, TDPEKA MED CENTER,6660A
233-9681
37 M 2501 62 DPH
DUNAGIN, JACK A, 918 W 10TH,6660A
233-9681
20 M 1902 4A P
DURST JR, RDBERT D, CDNTINENTAL MED BLDG,66606
357-5166
42 M 2803 69 D
ELDER, DDUGLAS M, 310 MED ARTS BLDG,66604
234-3A51
A1 M 1902 69 DR
FEAGAN, JERRY, 901 GARFIELD,66606
354-9591
39 M 1902 63 GE
FERNANDEZ, LUIS A, 1615 W 8TH,66606
233-8961
14 M 27501 41 PD
FIELD, RICHARD A, 303 MEDICAL ARTS BLDG,66604
235-3451
29 M 1902 55 ANES
FILLMAN, ELDDN M, 301 MEDICAL PLAZA BLDG,66604
233-4256
20 M 1611 AA U

FORD, FRED L, 220 MEDICAL ARTS BLDG,66604
234-5516
11 M 2501 36 GS
FOSTER, CHARLES G, 110 CONT MED BLDG,66606
232-6964
21 M 4102 47 IM
FOSTER, O BERNARD, 3111 JEWELL,66611
-
14 M 2501 38 NP
FUSILLO, MICHAEL, BOX 829,66601
234-9566
48 M 3503 74 P
GANDHI, SHANTIKUMAR K, 631 HORNE,66606
233-9636
40 M 49501 67 TS
GANZARAIN, RAMON C, MENNINGER FD,66601
234-9566
23 M 23101 47 P
GAY, JOHN D, 310 MED ARTS BLDG,66604
234-3451
42 M 4802 68 OR
GENDEL, JOSEPH E, 918 W 10TH,66604
233-9681
12 M 4804 37 ORS
GIESSEL, MICHAEL D, CONTINENTAL MED BLDG,66606
357-5166
48 M 1902 74 O
GIMPLE, KENNETH, 631 HORNE,66606
233-7491
45 M 1902 71 ORS
GLEASON, JIMMIE A, 800 LINCOLN,66606
233-5101
33 M 1902 58 OBG
GODFREY, KENNETH E, 3200 W 29TH TERRACE #7,66614
273-6153
15 M P
GOERING, EMIL L, 600 MADISON,66607
354-5100
27 M 1902 57 IM
GOOTEE, JOSEPH E, 3204 W 17TH,66604
-
21 M 2834 47 DO
GRAHAM JR, CHARLES P, 631 HORNE #400,66606
354-9504
40 M 3601 65 GS
GRAY, DAVID E, 115 W CRANE,66603
234-8621
16 M 1606 42 GEN
GRAYIB, ANTOINE S, MEMORIAL HOSP,66607
354-5100
18 M 60501 46 IM
GREENBERG, MARK, 310 MED ARTS BLDG,66604
234-3451
46 M 1611 72 R
GREENE, HORACE T, 1710 W 10TH,66604
354-7508
15 M 401 42 FP
GREENWOOD, EDWARD D, 3617 W 6TH,66601
234-9566
01 M 702 38 CHP
GREER, RICHARD H, 918 W 10TH,66604
233-9681
09 M 1902 39 IM
GRIGSBY JR, GEORGE T, 1931 GRANTVILLE RD,66619
233-9470
41 M 3601 66 IM
GUTOVITZ, ALLEN LOUIS, PROFESSIONAL BLDG STE 201,66604
233-9643
46 M 1611 72 CO
HACKER, ELAINE MARY, 3026 QUAIL CREEK,66614
296-3981
25 F 2604 50 OBG
HAINES, ROBERT A, 2700 SIXTH,66606
296-3436
16 M 3841 42 P
HALLEY, M MARTIN, 40 MED ARTS BLDG,66604
233-1710
27 M 2401 53 TS
HAMILTON, NORMAN G, BOX 829,66601
234-9566
51 M 1902 76 FP
HARRIS, HUBERT L, 210 MED ARTS BLDG,66604
233-3151
12 M 1803 39 D
HARRIS, PATRICIA A, 1617 W 26TH,66611
354-8461
29 F 1902 54 IM
HARRISON, HALL E, 901 GARFIELD,66606
354-9591
39 M 2802 65 IM
HARTOCOLLIS, PETER, MENNINGER FD,66601
234-9566
22 M 86905 55 P

HEBBAR, SATYA N, 1710 W TENTH SUITE 201,66604
233-9643
39 M 49509 62 CO
HERRERA, JORGE J, 2825 CALIFORNIA,66605
354-5100
27 M 64901 55 IM
HIEBERT, JOHN B, 901 GARFIELD,66606
354-9591
40 M 1902 68 CO
HILL, ROBERT N, 901 GARFIELD,66606
354-9591
14 M 1902 67 IM
HILST, WILBUR DEAN, 1271 WOODHULL,66614
273-5870
31 M 3005 55 GS
HIRSCHBERG, J COTTER, MENNINGER FD,66601
234-9566
15 M 1602 40 CHP
HISZCZYNSKYJ, ROMAN, 1500 W TENTH,66604
354-6870
35 M 1803 66 PATH
HOBBS, DONALD D, 200 CONTINENTAL MED BLDG,66606
233-7491
28 M 2401 54 ORS
HOHERZ, DAVID G, 314 MED ARTS BLDG,66604
235-1170
45 M 1902 76 TS
HORNE, JAMES B, 820 QUINCY STE 207,66604
234-2120
26 M 1606 52 P
HOSTETTER, JAMES P, 1500 W TENTH,66604
354-6100
43 M 1902 69 EM
HOYT, ARTHUR W, 2055 CLAY,66604
234-5663
14 M 2501 40 P
HSU, CHENG H, 1516 W SIXTH,66606
233-9681
41 M 38502 66 U
HSU, SHIN-FU, MEDICAL PLAZA BLDG,66604
232-0362
43 M 24402 68 OTO
HUAMAN, ANTONIO M, 631 HORNE HB-20A,66606
233-3566
29 M 73701 56 PATH
HUSTON, JOSEPH W, 918 W 10TH,66604
233-9681
35 M 1902 62 ORS
HUTTON, FREDERICK A, 102 MED PLAZA BLDG,66604
234-0553
29 M 6701 58 PS
ILORETA, ALFREDO TAB/TOL, VETERANS ADM MED CENTER,66622
272-3111
47 M 74801 71 U
JACKSON, LINDA H, 212 WOODLAWN AVE,66606
234-9566
42 F 3601 67 CHP
JACOBY II, ROBERT E, 340 CONT MED BLDG,66606
232-9394
46 M 2307 72 FP
JANSSEN, ERWIN T, MENNINGER FD,66601
234-9566
36 M 1803 62 P
JOHNSON, EDEN E, 5020 SW 28TH,66605
273-6700
39 F 74808 61 P
JOHNSON, FREDERICK E, 1108 1ST NATL BK TWR,66603
-
92 M 2843 21 DO
JOSEPH, BRIAN W, 130 CONT MED BLDG,66606
234-8653
38 M 35205 61 CHP
JOSS, CHARLES S, 221 MED ARTS BLDG W,66604
232-0444
14 M 1606 40 GS
JOYCE, G BERNARD, 200 CONTINENTAL MED BLDG,66606
233-7491
17 M 1902 44 ORS
JULIUS, SATISH CHANDRA, 2700 W SIXTH,66606
296-4348
40 M 49508 68 OPH
KATZ, JEROME B, MENNINGER FD,66601
234-9566
22 M 2101 44 P
KAVEL, KARL K, MED PLAZA BLDG,66604
234-2663
36 M 3605 64 PDA
KEARNS, NORBERT W, BOX 829,66601
234-9566
43 M 1002 70 P
KELLY, DAN A, 109 MED ARTS BLDG W,66604
232-8224
39 M 2803 64 PD

KENNEDY, HOWARD U. 1115 W TENTH SUITE A.66604
233-8268
18 M 401 44 IM
KEYS JR, ROBERT C. 303 MED ARTS BLDG.66604
235-3451
36 M 1902 62 ANES
KEYS SR, ROBERT C. 3034 LYDIA.66614
-
97 M 1902 27 OD
KIM, YONG W. CONT MED BLDG.66606
232-6964
28 M S8302 49 IM
KINDLING, PAUL H. 40 MED ARTS BLDG.66604
233-1710
30 M 3545 61 TS
KIRKEGAARD, ROGER S. 835 WESTERN.66606
233-6493
30 M 1803 56 DPH
KLEINHOLZ JR, EMIL JOHN. 600 MADISON.66607
354-5284
39 M 3503 65 IM
KLEMMER, HERBERT. MENNINGER FD.66601
234-9566
11 M 4102 37 P
KOVARIK, ERNEST O. 900 WASHBURN.66606
357-5171
36 M 3005 64 OPH
KROLL, HARRY G. 200 CONTINENTAL MED BLDG.66606
233-7491
24 M 1602 50 ORS
LAWSON, DWIGHT. 108 MEADOW LANE.66606
-
06 M 2802 30 IM
LAWSON, ROBERT C. 310 MED ARTS BLDG WEST.66604
234-3451
25 M 801 48 R
LEE, SONG OOW. 303 MED ARTS BLDG.66604
235-3451
43 M 38505 68 ANES
LEE, SONG PING. 918 W 10TH.66604
233-9681
34 M 38502 61 OTO
LEGLER, GARY LEE. 2835 SE SKYVIEW CT.66605
233-8961
50 M 2878 77 GP
LENTZ, WILLIAM R. 308 MED ARTS BLDG.66604
235-3443
24 M 1902 53 FP
LESSENDEN JR, C M. 103 MEDICAL PLAZA BLDG.66604
234-5533
18 M 1902 43 O
LEVY, EDWIN Z. 4125 SW GAGE CTR DR.66604
273-5610
29 M 1505 54 P
LIN, MAU SHONG. MEMORIAL HOSPITAL.66607
354-5100
45 M 24402 71 IM
LONG, JOHN W. 1001 HORNE ST.66604
233-1710
43 M 1902 70 TS
LUMB, RAYMOND C. 901 GARFIELD.66606
354-9591
42 M 1001 68 RHU
LYNCH, JOHN A. 300 CONT MED BLDG.66606
357-0301
30 M 2834 55 DRS
MAGILL, ALFRED MORTON. 3261-F RANDOLPH, 66611
233-9643
30 M 1642 58 CO
MARSHALL, B M. 1825 SW 34TH.66611
-
08 M 2802 34 U
MARTIN, WILLIAM O. 303 MED ARTS BLDG.66604
235-3451
19 M 1902 44 ANES
MAU, WALTER. 301 MEDICAL PLAZA BLDG.66604
233-4256
16 M 1611 40 U
MCCARTER, DUANE K. 2101 W 10TH.66604
233-8979
26 M 1902 58 IM
MCCLELLAN, JOHN W. 1710 W 10TH.66604
234-2624
11 M 3005 36 ENT
MCCLURE, JAMES A. 301 MED PLAZA BLDG.66604
233-4256
18 M 1902 44 U
MCELROY, ROBERT T. 221 MED ARTS BLDG.66604
232-0444
35 M 1902 61 GS

MCELROY, WILBUR J. 1616 W EIGHTH.66606
233-2280
35 M 1902 61 DPH
MEGIBOW, ALAN D. 918 W 10TH.66604
233-9681
37 M 3503 64 CHP
MEIDINGER, RICHARD. 310 MED ARTS BLDG.66604
234-3451
39 M 1902 65 OR
MENNINGER, KARL A. BOX 829.66601
-
93 M 2401 17 P
MENNINGER, ROBERT G. 3617 W 6TH ST.66601
234-9566
22 M 3545 52 P
MENNINGER, ROY W. 3617 W 6TH.66601
234-9566
26 M 3520 51 P
MENNINGER, W WALTER. THE MENNINGER FD.66601
234-9566
31 M 3520 57 P
MILLS JR, PHILIP E. 901 GARFIELD.66606
357-6171
36 M 1902 64 N
MITTLEMAN, FREDERICK S. MENNINGER FD.66601
234-9566
45 M 3005 70 P
MODLIN, HERBERT C. MENNINGER FD.66601
234-9565
13 M 3005 38 P
MOORE, HUGH C. 1500 WEST 10TH.66604
354-6870
33 M 4812 59 PATH
MORRIS, MERLE D. 1401 W 10TH.66604
234-2877
21 M 1902 45 DBG
*MORRISON, MICHAEL R, 8TH & LINCOLN, 66606
233-5101
50 M 1902 73 OBG
MORROW JR, J TARLTON. MENNINGER FD.66601
234-9566
23 M 4804 47 CHP
MOYER, JOHN D. 301 MEDICAL PLAZA BLDG.66604
233-4256
39 M 1902 65 U
MUELLER, ARNOLD V. 901 GARFIELD.66606
354-9591
31 M 3005 57 IM
MURPHY, THOMAS MEAD. PO BOX 829.66601
234-9566
41 M 3901 68 P
MYERS, JO ANN. MENNINGER FD.66601
234-9566
28 F 1902 53 P
NABOURS, RICHARD D. 4228 W 29TH ST TERR.66614
272-7190
27 M 1902 54 FP
NANCE, JOEL. MENNINGER FOUNDATION.66601
234-9566
42 M 3545 72 P
NELSON, JAMES N. 918 W 10TH ST.66604
233-9681
30 M 1902 59 P
NICE, G WILLIAM. 112 MED ARTS BLDG EAST.66604
235-8090
22 M 1902 46 IM
NOVOTNY, PETER C. MENNINGER FD.66601
234-9566
30 M 1S407 55 P
NYSTROM, CURTIS A. 2707 W 29TH.66614
272-8440
23 M 1902 54 FP
O'NEIL, ROBERT H. 901 GARFIELD.66606
354-9591
20 M 1902 45 IM
OBOURN, ROBERT L. 1150 OAKLEY.66604
234-9566
19 M 2802 50 P
PALMBERG, KENT E. 901 GARFIELD.66606
354-9591
49 M 1902 74 IM
PAPP, ROBERT. 3230 W 30TH.66614
272-5956
38 M 1611 63 FP
PARKS, GILBERT R. 529 QUINCY.66603
233-1785
44 M 4102 73 P
PARMAN, ROBERT O. 109 MED ARTS BLDG WEST.66604
232-8224
27 M 1902 54 PO
PARULKAR, DEEPAK S. ST FRANCIS HOSP & MED CTR.66606
295-8440
49 M 67 ANES

PATEL, VINOO, 655 WESTCHESTER RD,66606
3S7-6171
47 M 49531 69 N
PATINO, EDGAR, MENNINGER FD,66601
234-9S66
41 M 26406 66 P
PATRICK, FRED EDWARD, 109 MEDICAL ARTS BLDG,66604
232-8224
45 M 1902 71 PD
PAYNE, ROBERT R, 200 CONTINENTAL MED BLDG,66606
233-7491
29 M 1902 55 ORS
PENKA, WAYNE E, DEPT OF PATHOLOGY,66606
295-8472
46 M 3005 72 PATH
PENN, GEORGE M, VA MED CTR, 2200 GAGE, 66622
272-3111
30 M 4802 58 P
PETERSON, DEAN L, 303 MED ARTS BLDG WEST,66604
235-3451
24 M 1902 54 ANES
PETERSON, ROBERT L, STORMONT-VAIL EMERGENCY,66606
3S4-6108
36 M 1902 62 FP
PETERSON, VERNON J, 310 MED ARTS BLDG,66604
234-3451
42 M 512 68 R
PETRIK, EDWIN L, MEMORIAL HOSPITAL,66607
3S4-5100
35 M 1902 64 IM
PETTERSON, DENNIS CRAIG, 310 MEDICAL ARTS BLDG,66604
234-3451
49 M 1902 74 R
PFUETZE, ROBERT E, 209 MED ARTS BLDG EAST,66604
232-92S7
09 M 1902 35 OBG
PIERCE, CHARLES F, 918 W 10TH,66604
233-9681
24 M 4101 51 OBG
PIERCE, DONALD R, 307 MED ARTS BLDG EAST,66604
235-2226
23 M 5101 49 FP
POLLY, RICHARD E, 631 HORNE ST,66606
3S7-0301
42 M 1803 68 ORS
PORTER, ROBERT D, 901 GARFIELD,66606
3S4-9591
41 M 2802 67 IM
POWELL, WILLIAM R, 833 GARFIELD,66606
233-8941
30 M 1902 54 GS
POWELL II, BENSON M, 400 CONTINENTAL MED BLDG,66606
3S4-9S04
26 M 1606 49 TS
PRESTON, RALPH R, 1710 WEST 10TH,66604
234-2624
19 M 1902 44 OPH
PROKOP, BRADFORD S, 900 WASHBURN,66604
233-3900
32 M 1606 57 OPH
PYLE, LUCIEN R, 211 MED ARTS BLDG EAST,66604
233-1304
01 M 1601 28 OBG
RAINBOW-EARHART, KATHRYN A, 825 WESTERN,66606
233-1730
21 F 4707 48 P
RAMSEY, BARTLETT W, 109 MED ARTS BLDG,66604
232-8224
25 M 1902 50 PO
RANSDELL, EDGAR C, 800 LINCOLN,66606
233-5101
41 M 3005 66 OBG
RANSOM, JAMES H, 223 MEDICAL PLAZA BLDG,66604
234-2663
36 M 1803 62 A
REIMER, DONALD R, ANES DEPT ST FRANCIS HOSP,66606
3S4-8411
34 M 702 62 ANES
REINKING, VICTOR E, 918 WEST 10TH,66604
233-9681
26 M 1902 51 IM
REYMOND, RALPH D, S-F CAP REG RADIOTHER CTR,66606
234-3451
37 M 2301 67 R
RICHARDSON, J M, TOPEKA MEDICAL CENTER,66604
233-9681
47 M 1611 74 IM
RIEDEL, ROBERT H, 6731 SW ALYESBURY RD,66610
235-0011
04 M 2802 28 PH
ROBERTS, WARREN E, PO BOX 4047,66601
272-5797
25 M 1902 57 FP

ROBINSON, DAVID B, 800 LINCOLN,66606
233-5101
47 M 1902 73 OBG
ROCERETO, PAUL V, 1001 MULVANE,66604
233-0401
47 M 1902 73 FP
ROEDER, ROBERT E, 901 GARFIELD,66606
354-9591
40 M 1902 67 IM
ROSS, JACK L, MENNINGER FD,66601
234-9566
32 M 4812 56 P
ROTERT, LARRY, 301 MEDICAL PLAZA BLDG,66604
233-4256
38 M 3007 66 U
ROY, WILLIAM R, 1700 W SEVENTH,66606
295-8991
26 M 1606 48 ADM
RUCKER, CLEMENS, 1205 W 29TH,66611
86 M 2822 05 OO
RUNNELS, JOHN B, 901 GARFIELD,66606
3S7-6171
35 M 1902 61 NS
RUPP, RICHARD J, 901 GARFIELD,66606
354-9591
42 M 3841 68 CD
SANCHEZ, ROGELIO, 918 WEST 10TH,66604
233-9681
31 M 64901 64 U
SARGENT, JOSEPH D, MENNINGER FD,66601
234-9S66
32 M 2501 58 IM
SAYLOR, EDWARD H, 918 WEST 10TH,66604
233-9681
39 M 1902 65 PD
SAYLOR, LESLIE L, 918 WEST 10TH,66604
07 M 1606 35 DO
SAYLOR, MARK, 918 WEST 10TH,66604
233-9681
37 M 1902 66 GS
SCAMMAN, W WIKE, 115 W CRANE,66603
234-8621
32 M 4705 57 PATH
SCHLOESSER, HARVEY L, 918 MERCHANTS NATL BK,66612
235-3184
21 M 3901 51 P
SCHLOESSER, PATRICIA T, KANSAS ST BD OF HEALTH,66620
24 F 3901 49 PD
SCHRAM, PETER CHARLES, 130 CONTINENTAL BLDG,66606
234-8653
39 M 2507 69 P
SEGERSON, JOHN A, 901 GARFIELD,66606
3S7-6171
18 M 3545 43 N
SEHOEV, JOAN, MEMORIAL HOSP,66607
354-5100
40 F 6101 63 FP
SETTLE SR, RUSSELL O, 2320 CHELSEA DRIVE,66614
04 M 1902 29 OO
SEVIER, SAMUEL M, 3107 W 21ST,66604
296-5306
19 M 4812 44 IM
SHAW, JOSEPH L, MED ARTS BLDG,66604
235-6221
34 M 511 60 ORS
SHEAFOR, DOUGLAS, 918 W 10TH,66604
233-9681
34 M 1902 60 P
SHELTON, STEPHEN E, 918 W 10TH,66604
233-9681
35 M 702 61 P
SHERWOOD JR, CLARENCE E, CONTINENTAL MED BLDG,66606
3S4-9504
22 M 702 53 GS
SIMPSON, WILLIAM S, MENNINGER FOUNDATION,66601
234-9566
24 M 6001 48 P
SISK, PHILLIP B, 310 MED ARTS BLDG WEST,66604
234-3451
32 M 1803 56 R
SMITH, LEO A, 918 BUCHANAN,66604
08 M 3006 33 OO
SMITH, STEPHEN BUHLER, 901 GARFIELD,66606
3S4-9591
48 M 1902 74 IM
SNARR, JACK W, MED ARTS BLDG W,66604
234-34S1
41 M 6201 65 OR

SPEARMAN, JESSE L, MED ARTS BLDG RDDM 24,66604
234-2879
20 M 1902 54 DBG
SPENCER, MILLARD C, 310 MED ARTS BUILDING,66604
234-3451
28 M 1902 55 R
SPENCER, WAYNE E, 103 MED ARTS BLDG EAST,66604
233-9686
38 M 1902 64 IM
STEIN, JOSEPH M, 901 GARFIELD,66606
357-6171
24 M 3519 47 N
STOCK, KARL W, 1710 W 10TH,66604
235-5205
13 M 2834 37 OPH
SUFI, MOHAMMAD ASHRAF, MEMORIAL HOSP,66607
354-5286
43 M 70402 68 IM
SUFI, QAISER A, 115 W CRANE,66603
234-8621
44 F 70402 68 PATH
SUTTON, RICHARD D, 204 MED ARTS BLDG,66604
235-6221
38 M 4706 67 ORS
SWOGGER JR, GLENN, MENNINGER FD,66601
234-9566
35 M 3806 60 P
TAPPEN, DANIEL L, 800 LINCOLN,66606
233-5101
16 M 1902 41 OBG
TARGOWNIK, KARL K, 1218 W TENTH,66604
296-7281
15 M 40710 49 P
TARNOFF, GERALD M, BOX 829,66601
234-9566
48 M 73 P
TARNOWER, WILLIAM, MENNINGER CLINIC,66601
234-9566
21 M 4802 48 P
TEMPERO, STEPHEN J, 310 MED ARTS BLDG,66604
234-3451
42 M 1606 67 R
THOMS, NORMAN W, 40 MED ARTS BLDG,66604
233-1710
34 M 2501 59 TS
THURSTON, DAVID E, 200 CONTINENTAL BLDG,66606
233-7491
29 M 1902 55 ORS
TOTH, JOHN ROY, 631 HORNE - SUITE 340,66606
232-9394
49 M 1902 71 FP
TOZER, RICHARD C, 901 GARFIELD,66606
357-6171
19 M 4102 45 NS
TRAVIS, JOHN W, S-F CAP REG RADIOTHER CTR,66606
295-8000
29 M 1606 55 R
TREES, CLYDE B, 3700 HUNTOON,66604
—
09 M 2401 33 OD
TREGER, NEWMAN V, 1704 W 10TH,66604
354-8761
16 M 1902 40 IM
TRUITT, WILLIAM ROY, 1700 W TENTH,66606
354-8411
37 M 3515 66 EM
TWEMLOW, STUART W, 820 QUINCY - ROOM 418,66612
233-1607
41 M 67101 66 P
UHR, NATHANIEL, MENNINGER FOUNDATION,66601
234-9566
00 M 3519 21 IM
VANDE GARDE, LARRY D, 800 LINCOLN,66606
233-5101
41 M 1803 66 OBG
VISWANATHAN, BYRAVAN, 600 MADISON,66607
354-5100
39 M 49534 62 HEM
VOGEL, STANLEY J, 901 GARFIELD,66606
354-9591
44 M 2802 70 IM
VOTH, HAROLD M, MENNINGER FOUNDATION,66601
234-9566
22 M 1902 47 P
WALLACE, LEO F, 5500 W 24TH,66614
296-4424
17 M 1902 41 EM
WALLS, WILLIAM J, 310 MED ARTS BLDG,66604
354-6171
39 M 2834 66 OR
WALZ, ROYCE C, 1710 W 10TH SUITE 205,66604
234-2676
27 M 15407 60 P

WARD, HOWARD N, 901 GARFIELD,66606
354-9591
37 M 1606 62 HEM
WARE, LUCILE M, MENNINGER FOUNDATION,66601
234-9566
29 F 3501 53 P
WARRICK, DAVID ALAN, 918 W TENTH,66604
233-9681
49 M 3843 76 IM
WATERS, DALE A, 631 HORNE SUITE 428,66606
233-9636
41 M 5605 67 COS
WEAVER, WALTER D, 900 WASHBURN ST,66606
357-5171
41 M 1902 69 OPH
WEBER, DARRELL J, 1710 W 10TH,66604
233-2305
16 M 1902 44 FP
WHITE, HARRY H, 901 GARFIELD,66606
357-6171
34 M 1902 58 N
WIEDERMAN, FRANCIS JAMES, 3100 SE PISCES, 66605
—
46 M 74809 77 GP
WIGGLESWORTH, ANNE, 918 W TENTH,66604
233-9681
40 F 1902 75 OBG
WIKSTEN, VERNON C, 807 TERRACE AVE,66611
—
11 M 1902 37 OD
WILCOX, DONALD E, 6700 S TOPEKA AVE,66620
296-3782
24 M 1902 55 PH
WILDS, CHARLES E, 3227 MAC VICAR COURT,66611
—
M P
WILSON, MARVIN H, 38 MED ARTS BUILDING,66604
234-0591
38 M 1003 64 GS
WING, NANCY J, 700 HARRISON,66603
295-3175
30 F 5104 56 ADM
WONG, NORMUND, MENNINGER FD,66601
234-9566
34 M 502 59 P
*WOOD, EDWARD R, 901 GARFIELD, 66606
354-9591
49 M 1902 76 IM
WOODS, ROBERT P, 901 GARFIELD,66606
357-6171
14 M 6701 40 N
YOUNG, THEODORE E, 107 MED ARTS BLDG WEST,66604
232-0576
22 M 2307 46 PD
ZACHARIAS, DAVID LLOYD, 1500 W TENTH,66606
354-6870
26 M 1902 53 PATH
ZIMMERMAN, WILLIAM H, CONTINENTAL BLDG,66606
232-4377
20 M 3006 52 GS

TRIBUNE—316
(Southwest Kansas Society)

WERNER, WILLARD F, ,67879
376-4251
24 M 1902 52 FP

TROY—913
(Northeast Kansas Society)

MASTERSON, MELVIN LEROY, 210 S MAIN,66087
—
23 M 4901 48 R

ULYSSES—316
(Southwest Kansas Society)

BREWER, MARSHALL A, PO BOX 687,67880
356-1261
19 M 1902 46 FP
GREENWOOD, JAMES F, PO BOX 927,67880
356-1261
33 M 1611 65 FP
TILLOTSON, DON R, 223 N MAIN,67880
356-1261
32 M 1902 65 FP

VALLEY CENTER—316
(Sedgwick County Society)

WILSON, ROBERT L, RR 1,67147
685-2563
30 M 1902 57 EM

WAKEENEY—913
(Central Kansas Society)

BERNER, NEAL E, 323 RUSSELL,67672
743-2124
44 M 1902 72 FP
HAMILTON, JAMES J, MEDICAL CENTER,67672
743-2124
30 M 1902 55 FP

WAMEGO—913
(Pottawatomie County Society)

BORGENDALE, LLEWELLYN V, 1601 SUNSET,66547
456-2291
29 M 1902 60 FP
BRADEN, BILL L, 507 ELM,66547
456-2291
31 M 1902 60 FP
CLARK, LAURENCE A, 507 ELM,66547
456-2291
12 M 1902 42 FP

WASHINGTON—913
(Northeast Kansas Society)

BITZER, DONALD A, 115 W 3RD,66968
-
03 M 3005 26 OD
HODGSON, DAVID K, 107 E THIRD,66945
-
M FP

WATHENA—913
(Northeast Kansas Society)

PETERSON JR, EVAN A, 324 ST JOSEPH ST,66090
989-3122
24 M 1803 55 FP

WELLINGTON—316
(Tri-County Society)

ANDERSON, LARRY R, 1323 NORTH A,67152
326-3301
43 M 1902 73 FP
COLE, WARD M, 110 N JEFFERSON,67152
326-7221
08 M 1902 36 FP
DIACON, JAMES L, 1619 N KEYES,67152
326-2111
24 M 3901 52 FP
MCCORMICK, EUGENE C, SECURITY STATE BANK BLDG,67152
326-3914
31 M 1902 56 FP
MCGOVERN, JAMES L, 124 E LINCOLN,67152
326-2111
22 M 3901 52 FP
NALDOZA JR, FAUSTINO M, 1323 NORTH A,67152
326-8171
M 74801 64 GS
WEIGAND, JOEL T, 1323 NORTH A,67152
326-3301
43 M 1902 70 FP

WESTMORELAND—913
(Pottawatomie County Society)

DECHAIRO, THOMAS, DECHAIRO HOSP,66549
457-3311
13 M 1902 36 FP

WICHITA—316
(Sedgwick County Society)

ABBAS, DILAWER H, 1152 S CLIFTON,67218
685-1111
45 M 70402 71 N
ADAMS, AUSTIN J, 1056 KEVIN,67208
-
10 M 4804 36 OD
AGUSTIN, CONRADO M, 103S N EMPORIA STE 165,67214
267-3389
38 M 74807 62 OBG
AHLSTRAND, RICHARD A, 3333 E CENTRAL STE 214,67208
685-1291
41 M 3005 67 R
ALFONSO, MANUEL, 3244 E DOUGLAS,67208
689-9445
37 M 84710 66 ANES
ALMONTE, PRISCILLA C, 303 S HILLSIDE,67207
684-7251
44 F 74801 67 ANES
ALMONTE, RODOLFO O, 1431 BLUFFVIEW DR STE 217,67207
686-3791
39 M 74801 64 OBG
ANDERSON, DONALD S, 3333 E CENTRAL,67208
685-4389
42 M 1902 69 ANES
ANDERSON, EUGENE G, SUTTON PLACE,67202
265-8619
19 M 1902 44 OBG
ANDERSON, HARRY O, 1451S E PAWNEE,67230
-
08 M 1601 34 OD
ARGOSINO, RODOLFO, 1148 S HILLSIDE,67211
683-6506
40 M 74801 63 GS
ARTZ, TYRONE O, 902 N HILLSIDE,67214
689-9171
41 M 1803 67 ORS
ASHMORE, ARTHUR L, 5025 E KELLOGG,67218
-
05 M 1902 32 OD
AUNINS, JOHN, 4853 HEMLOCK,67216
524-6805
28 M 4706 56 FP
BAILEY, DONALD C, 3243 E MURDOCK,67208
685-1491
37 M 3901 65 ORS
BARBA, ESTRELLA G, 1035 N EMPORIA SUITE 280,67214
262-1853
41 F 74802 66 P
BARBA JR MD, ANTONIO P, 1035 N EMPORIA,67214
267-3389
34 M 74807 62 OBG
BARKER, BENJAMIN W, 1148 S HILLSIDE,67211
683-4647
18 M 1902 51 FP
BARNETT, ARNOLD M, 5111 E 21ST,67208
685-2561
32 M 83601 54 N
BARTLETT, WAYNE C, 13 HAMPTON RD,67207
689-9124
07 M 1601 31 GS
BASS II, ORAL E, 851 N HILLSIDE,67214
685-1371
40 M 2803 71 U
BATES, MICHAEL D, 2733 EAST CENTRAL,67214
685-1277
48 M 3005 74 ORG
BATTISTE, CYNTHIA ELAINE, 3333 E CENTRAL SUITE 533,67208
681-2021
47 F 1606 73 PD
BAUMAN, M LEON, 1629 UNIVERSITY,67213
-
01 M 1902 44 OD
BAUMANN, PAUL A, 3333 E CENTRAL,67208
685-1291
32 M 5605 57 R
BEAVER, JAMES L, 245 COURTLEIGH DR,67218
263-3262
09 M 2101 35 GS
BEBAK, DONALD M, 2322 E CENTRAL,67208
263-6186
32 M 3508 58 ANES
BECKER, KARL E, 275 S PERSHING,67218
686-3963
43 M 2307 69 ANES
BENZ, LAURIE J, VETERANS HOSP,67207
685-2221
39 F 4101 65 IM
BETHEL, CHANDLER S, 5107 E 21ST ST,67208
682-6559
34 M 1902 59 IM
BHARGAVA, BAIKUNTH N, 959 N EMPORIA,67214
267-8322
37 M 49530 63 U
BIERMANN, HENRY J, 425 E MURDOCK,67214
265-6287
27 M 3006 52 GS
BIERMANN, WILLIAM J, 1435 LIEUNETT,67203
-
04 M 3006 29 OD
BINYON, KERNIE W, 4618 E CENTRAL,67208
684-2819
24 M 1902 56 FP
BLAYLOCK, HOYT C, 835 N HILLSIDE,67214
685-4395
21 M 1902 45 O

8LOOD, MARY J. 647 N HILLSIDE,67214
682-4559
13 F 1902 49 PD
8LOUSTEIN, PAUL A. WESLEY MEDICAL CENTER,67214
685-2151
42 M 3519 67 PATH
8OLT, MICHAEL, ST FRANCIS HOSP,67214
268-5000
M
8OND, ROGER C. 3243 E MURDOCK,67208
684-0251
40 M 5605 67 CD
8OSILJEVAC, JOSEPH E. 948 N VASSAR,67208
685-2151
M
8DYD, Z REX, 120 S MAIZE RD #12,67209
262-6211
26 M 3005 52 FP
8OYLE, HUGH H. 424 N WOODLAWN,67208
686-2193
33 M 3806 60 PATH
8RAKE, DAVID, 3333 E CENTRAL,67208
685-1291
43 M 702 68 R
8RAUN, KENNETH, 1431 S BLUFFVIEW,67207
638-4688
47 M 3519 72 OPH
8RAUN, THOMAS G. 3243 E MURDOCK #601,67208
685-2377
35 M 6001 61 N
8RAUN III, WILLIAM T. 3333 E CENTRAL,67208
685-1291
37 M 2802 61 R
8RECKBILL, DAVID L. 3333 EAST CENTRAL #214,67208
685-1291
38 M 1902 64 R
8RINTON, E HOLMES, 3244 E DOUGLAS,67208
689-9124
46 M 2101 70 GS
8RINTON, EDWARD S. 329 NORTH TERRACE DR,67208
-
15 M 1611 41 OD
8RITO, RAUL E. 3243 E MURDOCK,67208
682-4523
32 M 31901 59 U
8ROSIUS, FRANK C. 3243 E MURDOCK,67208
684-0251
25 M 1902 49 IM
8ROWN, DAVID J. 425 EAST MURDOCK,67214
265-6287
45 M 1902 71 GS
8ROWN, ROBERT L. 5025 E KELLOGG,67218
682-1534
21 M 1902 49 FP
8ROWN, RONALD C. 3243 E MURDOCK,67208
685-8231
47 M 2803 73 FP
8ROWN, RONALD L. 303 S HILLSIDE,67211
684-7251
45 M 3901 71 ANES
8ROWN, VAL J. 1802 N HYDRAULIC,67214
265-1461
24 M 1003 47 FP
8ROWNING, WILLIAM H. 851 N HILLSIDE,67214
685-1371
16 M 1902 43 U
8RUNER JR, KENNETH W. ST FRANCIS HOSPITAL,67214
268-5414
44 M 2401 70 PATH
8UBECK, RALPH W. 3244 E DOUGLAS,67208
689-9396
36 M 1803 62 IM
8UCK JR, BEN H. 1152 S CLIFTON,67218
684-2081
17 M 2834 43 TS
8UCKRIDAN, LATIF, 303 S HILLSIDE,67211
684-7251
31 M 6501 62 ANES
8URNEY, WILLIAM W. 1755 N MADISON,67214
264-8311
17 M 1902 52 FP
8URNEY II, WILLIAM W. 1755 N MADISON,67214
264-8311
50 M 4707 76 IM
8URPEE, JAMES F. 851 N HILLSIDE,67214
685-1371
39 M 5605 66 U
8UTH, DENNIS K. 2916 EAST CENTRAL,67214
684-5243
45 M 1902 72 IM
8UTIN, J WALKER, 3244 E DOUGLAS,67208
689-9477
23 M 1902 47 IM

8YRNE, JAMES PERRY, 905 N EMPORIA,67214
263-0296
42 M 2101 68 TS
CALIENDO JR, DANIEL J. WESLEY MED CENTER,67214
685-2563
41 M 1902 67 EM
CAPPER, STANLEY L. 3244 E DOUGLAS,67208
689-9206
37 M 1803 67 D
CARREAU, ERNEST P. 5105 E 21ST,67208
682-0281
17 M 1902 44 GS
CARTER, MACK A. 3333 E CENTRAL,67208
684-5158
18 M 1902 50 OPH
CASEY, JOHN J. 3243 E MURDOCK,67208
685-2377
25 M 2604 49 NS
CAUBLE, WILBUR G. 1148 S HILLSIDE,67211
683-1681
12 M 2834 39 GS
CAWLEY, LEO P. WESLEY MED CENTER,67214
685-2151
22 M 3901 52 PATH
CHANG, FREDERIC C. 905 N EMPORIA,67214
263-0296
35 M 2401 59 GS
CHAPMAN, JAMES H. POST OFFICE BOX 11344,67211
265-1684
27 M 4706 63 R
CHARD, FREDERICK H. 3244 E DOUGLAS,67208
689-9129
15 M 5605 39 D
CHO, SECHIN, UKSM - WICHITA,67214
268-8302
47 M 58302 71 PD
CHRISTMAN JR, CARL, 3333 E CENTRAL STE 303,67208
685-0559
48 M 4802 74 DBG
CIFUENTES, RAUL F. UKSM - WICHITA,67214
688-2295
40 M 23101 65 PED
CLARK, COURTNEY, 303 S HILLSIDE,67218
684-7251
30 M 1902 56 ANES
CLEAVER, EDGAR M. 1900 E NINTH,67214
268-8391
26 M 3005 54 PH
CLIFTON, H DAVID, 3600 E HARRY,67218
685-1111
41 M 401 65 R
COHEN, JUSTIN THOMAS, 3333 E CENTRAL STE 1,67206
684-5158
47 M 2803 74 OPH
COHLMIA, JERRY B. 1035 N EMPORIA,67214
263-5891
43 M 1902 70 IM
COLEMAN, THOMAS J. 959 N EMPORIA,67214
265-0749
18 M 3545 51 IM
COLLIER, HAROLD W. 303 SOUTH HILLSIDE,67211
684-7251
45 M 1902 71 ANES
CONCEPCION JR, EUGENIO S. 1152 SOUTH CLIFTON,67218
681-2401
39 M 74802 64 CD
CONRARDY, PETER A. 967 BACK BAY BLVD,67203
686-7327
42 M 502 69 ANES
COOK, DONALD RAY, 315 N HILLSIDE,67214
686-3391
42 M 2012 71 FP
COOK, G EDWARD, ST JOSEPH HOSPITAL,67218
685-1111
42 M 401 67 R
CORRIGAN, GEORGE F. 1828 W 18TH,67203
-
95 M 3005 20 DD
COSSMAN, F PRICE, 851 N HILLSIDE,67214
685-1371
28 M 1902 57 U
COWLES, GORDON T. 3333 E CENTRAL,67208
683-2661
32 M 1902 58 OBG
CRANE, DAVID D. 929 N ST FRANCIS,67214
262-6211
34 M 2501 60 PATH
CRISWELL, MILDRED LOUISE, 1520 SOUTH CLIFTON,67218
685-2371
42 F 1902 67 EM
CRONIN, DONALD J. 3244 E DOUGLAS,67208
689-9227
16 M 2604 40 ENT

CROW, ERNEST W, 3243 E MURDOCK,67208
684-0252
20 M 1902 44 CO
CROWLEY, EDWARD X, 345 N HILLSIDE,67214
682-4519
14 M 1643 39 GYN
CUMMINGS, RICHARD J, 427 N HILLSIDE,67214
686-6608
32 M 1902 57 OTO
DANIELS, ROBERT M, 929 N ST FRANCIS,67214
262-6211
24 M 1902 54 FP
DAVIDSON, HARRY T, 556 N BROADVIEW,67208
-
87 M 3802 11 OO
DAVIS, PAUL H, 315 N HILLSIDE,67214
685-8231
47 M 3901 72 FP
DAVIS, RONALD B, 1148 S HILLSIDE,67211
685-2152
46 M 1902 72 FP
DAY, HOWARD, 1035 N EMPORIA -SUITE 235,67214
263-5891
48 M 1902 74 NEP
DE BAKKER, JAN B, 1035 N EMPORIA,67214
263-4903
25 M 5104 59 GS
DECKERT, ROSALIE E, 3244 EAST DOUGLAS,67208
689-9212
47 F 2803 73 IM
DEJONG, DAVID C, 550 WEST SHORE DR,67209
268-5424
33 M 2501 59 PATH
DEMOSS, ELEANOR P, 3333 E CENTRAL SUITE 407,67208
682-5591
42 F 74802 66 PD
DOLAN JR, PHILIP JARVIS, 3244 E DOUGLAS,67208
689-9477
47 M 2101 73 GE
DONATELLE, EDWARD P, 1122 N TOPEKA,67206
268-5994
22 M 2504 50 FP
DONNELL, JAMES M, 3306 E CENTRAL,67208
682-6121
28 M 1902 55 FP
DOORNBOS, J FRED, 929 N ST FRANCIS,67214
262-6211
28 M 1902 57 R
DOWNING, GREGORY, 1123 NORTH OLIVER,67208
685-8499
M 1902
DRAKE, RALPH L, 4422 E 3RD,67208
-
99 M 4102 26 OO
DREVETS, CURTIS C, 3244 E DOUGLAS,67208
689-9178
30 M 1902 56 IM
DRIGGS, GUY K, ST FRANCIS HOSP,67214
268-5000
M
DUICK, GREGORY, 1035 NORTH EMPORIA #130,67214
263-3271
46 M 1643 72 CD
DURAND, ANTONIO C, 959 N EMPORIA,67214
263-7893
29 M 74807 56 U
DWORZACK, DAVID L, 1001 N MINNEAPOLIS,67214
268-8378
48 M 1902 73 IM
DYCK, CORA E, 702 WAVERLY,67218
-
00 F 1902 26 OO
DYER, VERNON E, 3244 EAST DOUGLAS,67208
689-9234
36 M 301 72 OBG
ECKERT, WILLIAM G, ST FRANCIS LAB,67214
262-6211
26 M 3519 52 PATH
EDWARDS, MANIS C, 3333 E CENTRAL,67208
683-2661
33 M 3005 58 OBG
EFTEKHARI, SAIED, 4016 E CENTRAL,67208
-
M
EGELHOF, RICHARD H, 925 NORTH EMPORIA,67214
263-1299
45 M 1902 73 FP
ELLIS, HARVEY D, 6511 E CENTRAL,67206
683-1022
24 M 1902 55 GS
ELNEN, WALTER T, 450 N TERR DR,67208
682-5671
03 M 1643 32 GS

EMERY, FRANK A, 10 ST JAMES PLACE,67206
-
05 M 2802 29 OO
EPLEE, JOHN P, ST JOSEPH MED CENTER,67218
685-1111
53 M
ERKEN, RONALD V, WICHITA PSYCHIATRIC CTR,67208
684-0201
29 M 2834 56 P
EVANS, FARRIS D, 521 RUTLAND RD,67206
-
05 M 1902 32 FP
EVANS, GRANT E, 4618 E CENTRAL,67208
682-6556
21 M 4901 46 FP
EVANS, ROGER WILLIAMS, 925 N EMPORIA SUITE A,67214
263-5889
39 M 1902 64 CO
EYSTER, ROBERT L, 3243 E MURDOCK,67208
685-1491
47 M 3901 73 ORS
FARHA, GEORGE J, 905 N EMPORIA,67214
263-0296
27 M 2101 57 GS
FARHA, S JIM, 905 N EMPORIA,67214
263-0296
31 M 1001 57 TS
FENDER JR, THOMAS H, 9400 E LINCOLN APT 113,67207
267-8439
25 M 4812 54 OS
FERRIS, BRUCE G, 825 N EMPORIA,67214
262-3495
43 M 1902 69 PS
FEUILLE JR, EDMOND G, 345 N HILLSIDE,67214
682-4572
50 M 4802 75 OBG
FIELDS, STEPHEN A, 7200 W 13TH,67230
722-4258
42 M 2878 72 FP
FINLEY, DENNIS R, 1035 N EMPORIA,67214
262-7429
36 M 1606 62 ORS
FISHER, JAMES B, 141 S OLD MANOR,67218
-
09 M 1902 36 OO
FITZGERALD, EDWARD J, 3600 E HARRY,67218
685-1111
22 M 3006 50 R
FITZIG, SANFORD, 3244 E DOUGLAS,67208
689-9344
46 M 4102 72 U
FLEMING, FORNEY W, 3243 E MURDOCK #200,67208
685-1491
43 M 4802 69 ORS
FLOWERS JR, CLELL B, 855 N HILLSIDE,67214
685-1381
22 M 1902 55 FP
FORD, CHARLES R, 1301 N WEST ST,67203
942-4279
38 M 1902 63 OPH
FOWLER, ROBERT J, 3244 E DOUGLAS,67208
689-9236
37 M 2802 63 IM
FRANCIS, NORTON L, 5205 E 21ST,67208
684-2838
10 M 3005 35 ENT
FRITZ, GEORGE E, WESLEY MED CENTER,67214
685-2151
18 M 2507 43 PATH
FRITZEMEIER, WILLIAM H, 835 N HILLSIDE,67214
685-4395
14 M 1902 41 D
FROMM, ARTHUR H, 315 N HILLSIDE,67214
685-2281
37 M 1902 63 FP
FULTON, JOHN K, 3333 E CENTRAL,67208
686-0732
18 M 5605 43 PUD
GALICHIA, JOSEPH P, PO BOX 594,67201
263-3271
42 M 1902 69 CD
GALVAN, ALONSO, 3243 E MURDOCK,67208
684-0251
38 M 64906 64 IM
GENGELBACH, RICHARD W, WESLEY MED CENTER,67214
685-2371
M
GENILO, AMANCIO C, 120 LONGFORD CT,67206
685-2371
37 M 74801 61 EM
GENILO, CELESTE A, 3244 EAST DOUGLAS,67208
689-9445
39 F 74801 62 ANES

GEORGE, EARL F, 406 E CENTRAL,67202
265-6991
35 M 1902 65 FP
GEORGE, M DON, PO BOX 8037, 67208
268-8388
31 M 1001 S6 P
GERBER, ALLEN D, S105 E 21ST,67208
682-0281
48 M 1902 71 GS
GESSLER, DONALD J, 1122 N TOPEKA,67214
265-2876
41 M 1902 67 FP
GILMARTIN, RICHARD C, 3243 E MURDOCK #601,67208
685-2377
32 M 4101 58 PDN
GIVNER, DAVID, 611 N HILLSIDE,67214
684-0501
03 M 2301 29 IM
GOERING, ROBERT C, 3600 E HARRY,67218
685-1111
25 M 64901 56 PATH
GOHIL, MAHENDRA N, 1823 RUTLAND,67206
684-0333
28 M 40930 66 DR
GOLDBERG, HERBERT R, 400 N WOODLAWN,67208
685-4215
33 M 3508 59 PD
GONZALEZ, FRANCISCO, 1035 NORTH EMPORIA #265,67214
265-3226
46 M 73706 70 IM
GONZALEZ, HIRAM, 1035 N EMPORIA,67214
262-1853
20 M 64901 52 P
GOODPASTURE, HEWITT C, 1035 N EMPORIA SUITE 105,67214
262-6211
43 M 1902 69 IM
GOULDNER, RENE M, 139 COURTLEIGH DR,67218
-
88 M 3501 18 OD
GOYLE, KRISHAN K, 1144 N ST FRANCIS,67214
267-0159
34 M 49529 63 CD
GOYLE, VIMAL, 1150 N ST FRANCIS,67214
267-9906
41 F 49529 65 OBG
GRAUEL, CHARLES W, 8310 CHALET,67207
262-1127
44 M 1902 70 ANES
GRAVES, JACK W, 3244 E DOUGLAS,67208
689-9124
17 M 1902 42 GS
GRAY, C LUCIEN, 4821 E CENTRAL,67208
684-5171
21 M 1902 45 ENT
GRAY, H TOM, 3244 E DOUGLAS,67208
689-9152
19 M 401 44 D
GREER, JAMES A, 3244 EAST DOUGLAS,67208
689-9227
43 M 1611 69 DTD
GRIBBLE, ROBERT N, BOX S94,67201
264-2401
43 M 1902 69 R
GRILLOT, FLOYD B, 814 S WOODLAWN,67218
684-0243
18 M 1902 51 FP
GROHS, HEINZ K, 550 NORTH HILLSIDE,67214
685-2151
42 M 15407 66 PATH
GRUND, FRANK M, 3244 EAST DOUGLAS,67208
689-9420
47 M 1803 72 IM
GRUSHNYS, ARNOLD, 3244 E DOUGLAS,67208
689-9445
19 M 40721 59 ANES
GSELL, GEORGE F, 3244 E DOUGLAS,67208
689-9316
07 M 1601 33 DPH
GUNTHER, LORETTA ANN, 550 N HILLSIDE,67214
685-2563
39 F 4107 65 GS
GUTHRIE, RICHARD A, 1001 N MINNEAPOLIS,67214
268-8228
35 M 2803 60 PD
HAGAN, C THOMAS, 959 N EMPORIA,67214
265-0789
16 M 3006 42 IM
HAGAN, FRANCIS J, PO BOX 1837,67201
262-1057
13 M 3006 39 FP
HALE, RALPH, 847 S HILLSIDE,67211
684-0295
18 M 1902 46 A

HALL, J ROGER, 3333 E CENTRAL,67208
685-5227
42 M 4802 68 DPH
HALPIN, EDWARD D, 1148 S HILLSIDE,67211
685-9229
33 M 1902 62 OBG
HARMS, EDWIN M, 5623 POLO DR,67208
-
06 M 3901 34 DO
HARRIS, FRANK H, 1035 N EMPORIA,67214
262-1853
09 M 1001 39 NP
HARRISON, A BROOKS, 1040 STRATFORD,67206
-
99 M 1902 25 DO
HARRISON, PAUL BARRY, 3243 EAST MURDOCK,67208
685-6222
49 M 1902 74 GS
HART, DILLIS L, 1152 S CLIFTON, 67218
684-2081
36 M 3901 64 GS
HARVEY, ROSEMARY B, 2004 WELLINGTON PL,67203
265-5674
24 F 1902 49 ADM
HATTRUP, RICHARD J, 610 N TYLER RD,67212
681-3016
31 M 3006 57 FP
HAWLEY, RAYMOND G, ST JOSEPH HOSP,67218
685-1111
39 M 1902 65 PATH
HAYES, WILLIAM L, 3243 E MURDOCK,67208
684-0251
28 M 1902 53 CD
HAYS, THOMAS H, 315 NORTH HILLSIDE,67206
681-1841
49 M 1902 75 FP
HENNING, CHARLES E, 320 N HILLSIDE,67214
682-3221
37 M 1902 63 ORS
HENSLEY JR, CLINE O, 3244 E DOUGLAS,67208
689-9272
20 M 1902 44 ORS
HERED, JOHN, 959 N EMPORIA,67214
262-3613
41 M 2802 67 N
HERSHORN, SIMON E, 3333 E CENTRAL,67208
685-1291
22 M 1902 46 R
HIEBERT, ABRAHAM E, 1530 W 13,67203
-
94 M 2802 25 DO
HINSHAW, ALFRED H, 6110 ONEIDA,67208
-
07 M 1902 33 DO
HINSHAW, CHARLES T, 256 N BLECKLEY DR,67208
682-8651
99 M 4705 26 PD
HIRATZKA, TOMIHARU, 6 ST CLOUD PLACE,67230
685-2151
13 M 511 43 PATH
HIZON, RAMON R, ST FRANCIS HOSP,67214
262-6211
38 M 74801 62 DR
HODGSON, ROBERT B, 841 NORTH BROADWAY,67214
263-6131
22 M 1902 47 FP
HODSON, HERVEY R, 1122 S CLIFTON,67218
-
03 M 1606 31 DO
HOLDEN, DAVID M, 3243 E MURDOCK SUITE 300,67208
688-3070
38 M 801 63 FP
HOLDEN JR, RAYMOND F, 262 SOUTH BROOKSIDE,67208
-
10 M 2802 33 DO
HOLLADAY, HARMON M, 327 N RUTAN,67208
681-0431
23 M 1902 52 ANES
HOLMES, JED, ST JOSEPH MED CTR - FP,67218
685-1111
M
HOROELT, DOUGLAS V, 3333 E CENTRAL,67208
682-6511
47 M 4802 72 OBG
HOUSHOLDER, DANIEL FAIR, 7705 KILLARNEY CT,67206
681-1795
43 M 1902 70 NM
HOUSHOLDER, MARTHA S, 835 NORTH HILLSIDE,67214
685-4395
46 F 1902 72 D
HOWARD, DONALD D, 959 N EMPORIA,67214
265-7241
11 M 1902 38 DPH

HUEBERT, DEAN A, 5025 E KELLOGG,67218
682-1534
22 M 1902 46 FP
HULL, KENNETH L, 1035 N EMPORIA,67214
265-7903
38 M 2301 69 P
HULTGREN, MYRON K, 855 N HILLSIDE,67214
685-1381
41 M 1902 68 FP
HUME, JOSEPH W, 3243 E MURDOCK,67208
685-2223
38 M 1902 69 OBG
HUMMER, LLOYD M, 3244 E DOUGLAS,67208
689-9323
32 M 3901 57 IM
HUSTEAD, ROBERT F, 3243 E MURDOCK,67208
681-0431
28 M 801 54 ANES
HYNES, HARRY E, 1035 N EMPORIA,67214
262-4467
35 M 53902 58 HEM
IBARRA, J LUIS, 1035 N EMPORIA,67214
262-1853
20 M 64901 46 P
IOBEIS, BAOR, 1144 N ST FRANCIS,67214
267-0159
47 M 87501 72 TS
IOANNOU, NICHOLOAS, 1111 N ST FRANCIS,67214
265-2613
48 M 41801 72 TS
JACKSON, CHARLES R, 1035 N EMPORIA,67214
263-0812
27 M 1606 53 GS
JAMES, VERNON L, 400 N WOODLAWN,67208
268-8302
29 M 3601 55 PD
JAZAYERLI, NABIL, 1152 S CLIFTON,67218
681-2401
44 M 87501 70 CD
JEHAN, SAYED S, MENTAL HEALTH CLINIC,67214
268-8251
33 M 70403 59 P
JENNEY, CHARLES B, 905 N EMPORIA,67214
263-2795
34 M 2834 61 GS
JESTER, SHELBY L, 250 N PERSHING,67208
686-7327
43 F 4102 74 ANES
JOHNSON, GARY L, 1123 INVERNESS DR,67218
M
JOHNSON, GEORGE K, UKSM WICHITA,67214
268-5984
40 M 1205 67 IM
JOHNSON, THOMAS E, 3333 E CENTRAL,67208
685-1291
41 M 1643 67 R
JONES, CLIFFORD E, 1148 S HILLSIDE,67211
685-3354
22 M 1902 50 FP
JUDILLA JR, FRANCISCO, 2322 EAST CENTRAL,67214
263-6186
44 M 74801 71 ANES
KADISON, HERBERT I, ST FRANCIS HOSP,67214
262-6211
44 M 1611 69 R
KARDATZKE, E STANLEY, 1301 N WEST STREET,67203
943-9342
39 M 1720 64 FP
KARDATZKE, JON K, 10300 MAPLE,67209
943-3271
36 M 1720 62 FP
KASHA, ROBERT L, 5025 E KELLOGG,67218
682-1534
11 M 2834 38 GS
KASSEBAUM, KENNETH G, 3420 EAST DOUGLAS,67208
685-6381
M 1606 60 CHP
KAUFMAN, EUGENE E, 3243 E MURDOCK,67208
685-1491
30 M 1902 56 ORS
KEENE, GEORGE H, 5025 E KELLOGG,67218
682-1534
20 M 1902 49 GS
KEENY, M GARY, 841 N BROADWAY,67214
263-6131
36 M 1902 66 FP
KELLER, JAMES P, 1431 SOUTH BLUFFVIEW,67218
685-1284
48 M 1902 74 IM
KELLY, ROBERT W, 3243 E MURDOCK SUITE 605,67208
682-6511
46 M 4802 72 OBG

KENDALL, TOM E, 825 N EMPORIA,67214
262-3495
37 M 3901 62 PS
KENDRICK, J GILLERAN, 3333 E CENTRAL,67208
682-6511
20 M 1902 46 OBG
KENNEDY, GERALD T, 16069 CAVALCADE LANE,67230
684-5243
35 M 1902 61 GE
KHICHA, GYANCHAND J, 905 N EMPORIA,67214
263-0296
37 M 49530 61 TS
KHOURY, GEORGE H, 3333 E CENTRAL,67208
681-2021
32 M 33002 55 PD
KIM, PAIK N, 1035 N EMPORIA,67214
262-4467
33 M 58302 58 HEM
KIMBLE, JAMES A, 3244 E DOUGLAS,67208
689-9316
45 M 702 71 OPH
KIRK JR, E DAVID, 1431 S BLUFFVIEW DRIVE,67218
685-1351
34 M 1902 62 IM
KISER, JOHN L, 3243 E MURDOCK,67208
685-6222
37 M 2802 62 GS
KISER, WILLARD J, 3243 E MURDOCK,67208
685-6222
05 M 4705 30 GS
KITCHEN, ROBERT R, 3420 E DOUGLAS,67208
685-2355
26 M 1902 52 CHP
KLONTZ, WILLIAM JOSEPH, UKSM WICHITA,67214
268-8388
51 M 1902 76 P
KNAPP, LESLIE E, 302 S CRESTWAY,67218
96 M 1902 25 OD
KNAPP, M ROBERT, 37 VIA ROMA,67230
684-7251
23 M 3519 47 ANES
KNAPPENBERGER, ROY C, 3244 E DOUGLAS,67208
689-9327
16 M 1902 41 PD
KNEIDEL, THOMAS W, 732 N TOPEKA, 67214
262-3476
40 M 4101 66 ORS
KOURI, SAMMY H, 3243 E MURDOCK,67208
682-2911
33 M 3901 57 GS
KRAUSE, ROLAND L, 855 N HILLSIDE,67214
685-1381
25 M 1902 53 IM
KRUPKA, JOHN J, 959 N EMPORIA,67070
262-3613
47 M 1642 73 NS
KUBINA, GLENN RICHARD, 5205 E 21ST,67208
684-2838
47 M 3840 72 OTO
KURTH, C JOSEPH, 925 N EMPORIA,67214
263-6177
10 M 3006 35 NP
KUTILEK, FRANK J, 7200 W 13TH ST SUITE 3,67212
722-4258
30 M 1902 57 FP
LACEY, STEFFAN R, ST FRANCIS HOSP,67214
268-5000
M 1902
LAI, JENG Y, 1111 N ST FRANCIS,67214
265-2613
41 M 38502 67 TS
LAMBERT, EUGENE KENT, 3244 E DOUGLAS,67208
689-9214
44 M 1611 70 IM
LANCE JR, JOHN F, 3244 E DOUGLAS,67208
689-9438
20 M 1902 45 ORS
LATIMER, KATHERINE, 3243 EAST MURDOCK,67208
686-7327
49 F 1205 75 ANES
LAUVER, MARY ANN, 1001 N MINNEAPOLIS,67214
268-8302
40 F 1902 74 PD
LAWN, CLAUDIA A, MID KS RADIOLOGY GROUP,67211
265-1684
50 F 1902 75 R
LAWN, RAYMOND A, 715 N MISSION RD,67206
683-8991
09 M 2604 35 AM
LAZAR, HARRY, 611 N HILLSIDE,67214
684-0501
08 M 1611 35 A

LEE, R REX, 6155 E HARRY,67218
685-2306
29 M 3901 5S FP
LEE JR, EDWARD S, 2002 E 17TH ST,67214
264-8273
09 M 4707 37 FP
LEISY, JERALD W, 3310 E DOUGLAS SUITE 101,67208
681-2937
42 M 1902 68 P
LEVINE, WILLIAM R, UKSM-WICHITA,67214
268-8388
42 M 1902 67 P
LIES, JAMES E, 3244 E DOUGLAS,67208
689-9240
43 M 1902 69 IM
LIES, RICHARD B, 3244 E DOUGLAS,67208
689-1111
42 M 1902 68 RHU
LIN, JOE J, 8630 HUNTINGTON,67206
268-5420
42 M 24404 69 PATH
LINDLEY, MILTON E, 657 S TERRACE DR,67218
-
14 M 1902 50 DO
LINHAROT, RONALD O, 3243 E MURDOCK,67208
683-2655
36 M 2803 64 OBG
LITTLE, L GILBERT, 122 S WESTFIELD AVE,67209
263-7614
99 M 401 27 P
LOCKHART, JOSEPH G, 5900 E CENTRAL,67208
684-7239
17 M 4113 43 PD
LOEFFLER, JAMES A, 400 N WOODLAWN,67206
685-5375
36 M 3841 63 A
LOEWEN, HENRY H, 2142 W 17TH,67203
-
03 M 1902 35 DD
LOEWEN, WILLIAM C, 10300 W MAPLE,67209
943-3253
41 M 1902 71 FP
LOGAN, GEOFFREY G, 345 N HILLSIDE,67214
682-4572
31 M 14303 56 OBG
LORCH, VICHIEN, UKSM-WICHITA,67214
268-8302
40 M 89101 65 PD
LOVETT, PAUL A, 110 PATTON,67208
683-6683
09 M 1902 45 ORS
LOW, HAROLD L, 201 S PERSHING,67218
684-2858
18 M 1902 44 FP
LUEKEN, LUEKE B, 3244 E DOUGLAS,67208
689-9234
23 M 40723 52 D8G
LUELLEN, THOMAS J, 3244 E DOUGLAS,67208
689-9244
17 M 1902 41 IM
LUZZATI, ENZO F, ST FRANCIS HOSPITAL,67214
262-6211
25 M 56119 50 R
MADISON JR, WARD N, 3600 E HARRY,67218
686-9432
37 M 3601 62 PATH
MAGIOSON, ELLIOT ARTHUR, 424 N WOODLAWN,67208
686-2193
43 M 1611 68 PATH
MAGSALIN, ROMULO, ST JOSEPH MED CENTER,67218
685-1111
M
MALIK, MUMTAZ ILAHI, 1035 N EMPORIA 290,67214
265-1631
47 M 70401 70 ORS
MANNING, ROBERT T, 1001 N MINNEAPOLIS,67214
268-8378
27 M 1902 54 IM
MANSOUR, BADIE S, 3243 E MURDOCK S 101,67208
686-7327
45 M 33002 69 ANES
MARSH, HENRY O, 925 N EMPORIA,67214
262-7598
18 M 1611 43 ORS
MARTIN JR, GLEN E, 2322 E CENTRAL,67214
263-6186
20 M 1902 49 ANES
MARYMONT JR, JESSE H, WESLEY MED CENTER,67214
685-2151
28 M 3515 54 PATH
MASTIO JR, GEORGE J, 3243 E MURDOCK,67208
684-5235
25 M 1902 52 GS

MATASSARIN, BENJAMIN M, 2916 E CENTRAL,67214
684-5243
20 M 1902 45 IM
MATASSARIN, FREDERICK W, 734 N EMPORIA,67214
265-2382
15 M 1902 37 U
MAWDSLEY, MICHAEL W, 3333 EAST CENTRAL #610,67208
686-6659
49 M 1902 74 PD
MCBRIDE, ANN, ST JOSEPH MED CENTER,67218
685-1111
F
MCCLANAHAN, WARD A, 5105 E 21ST,67208
684-8211
22 M 3005 48 ORS
MCCLELLAN, ERNEST L, 3243 E MURDOCK,67208
686-7327
38 M 4802 70 ANES
MCCOY, CHARLES P, 3333 E CENTRAL,67208
682-6511
17 M 3006 42 D8G
MCGUIRE, WILLIAM F, 3333 E CENTRAL,67208
683-5655
17 M 4101 43 PD
MCMAHON, MERRI M, 1001 NORTH MINNEAPOLIS,67214
268-8378
46 F 5404 72 IM
MCREYNOLDS, CHARLES R, WESLEY MED CENTER,67214
688-2899
21 M 3840 47 PATH
MEADOWS, DONALD C, 3333 E CENTRAL,67208
683-5611
36 M 102 69 OPH
MEEKER II, BRUCE P, 345 N HILLSIDE,67214
686-3384
30 M 1902 58 D8G
MEHRA, PROMILLA, 2810 EAST 21ST STREET,67214
681-1901
40 F 63 FP
MELEAN, JAIME, 1152 SOUTH CLIFTON,67208
681-2401
40 M 65 CO
MENAKER, JEROME S, 2703 E CENTRAL,67214
685-1227
16 M 1002 41 D8G
MENDIONES, L MARLENE, 835 N HILLSIDE,67214
685-4395
45 F 1611 70 O
MENDIONES, RUPERTO D, 3243 E MURDOCK -SUITE 404,67208
682-6585
44 M 1611 71 IM
MENEHAN, H JAMES, 3244 E DOUGLAS,67208
689-9404
26 M 1902 53 PD
MENKING, F W MANFRED, 3244 E DOUGLAS,67208
689-9336
34 M 40715 61 PD
MENKING, SUSAN MARGARET, UKSM WICHITA,67214
268-8302
41 F 3840 67 PD
MERCADER, MARIO S, 2322 E CENTRAL,67214
263-6186
43 M 74801 64 ANES
MEREDITH, W TOM, 1035 N EMPORIA,67214
263-7285
35 M 4812 61 IM
MERRITT, JOE P, 3243 E MURDOCK,67208
688-5211
46 M 2802 71 OBG
MERSHON, JAMES C, 925 N EMPORIA - SUITE A,67214
263-3271
37 M 1803 63 IM
MEULBRDEK, HARVEY J, 3244 E DOUGLAS,67208
689-9156
31 M 5605 56 A
MEYER, WARREN E, 1144 S CLIFTON,67218
684-5237
27 M 1606 51 GS
MICHELBACH, ALBERT P, 2916 E CENTRAL,67214
684-5243
35 M 2101 61 IM
MILFELD, DOUGLAS J, 905 N EMPORIA,67214
263-0296
45 M 4804 72 TS
MILLER, CLYDE W, 7618 DONEGAL,67206
-
09 M 2002 36 DO
MILLER, DAVID PATERSON, 3243 E MURDOCK SUITE 303,67208
688-3070
50 M 2803 77 FP
MILLER, DON E, 414S E KELLOGG,67218
682-6551
20 M 2802 44 GS

MILLER, PHILIP A. 1152 S CLIFTON,67218
684-2081
44 M 1902 70 GS
MILLS, CHARLES D. 1144 S WATER,67213
-
89 M 2002 14 OO
MILLS, GEORGE QUINTON, S50 N HILLSIDE,67214
-
48 M 3006 73 PATH
MIRZA, MEDO, 3333 E CENTRAL,67208
686-6683
38 M 40733 64 PDS
MOORE, DENNIS F. 1035 N EMPORIA,67214
265-3226
36 M 2101 62 HEM
MORGAN, DICK A. 3243 E MURDOCK,67208
686-7327
43 M 3901 69 ANES
MORGAN, JAMES I. 3124 S SENECA,67217
S22-2266
29 M 1606 53 FP
MORGAN III, LOUIS S. 8030 E KELLOGG,67207
683-3811
22 M 3901 48 FP
MORROW, THOMAS F. 3310 E OOUGALS,67208
685-1443
21 M S606 46 P
MOSELEY, JACK E. 1120 S CLIFTON,67218
682-4982
25 M 3901 53 FP
MOSIER, STANLEY JAY, 3243 E MURDOCK,67208
685-8231
42 M 1902 68 FP
MUELLER, VERNETTE A. 1431 S BLUFFVIEW,67218
684-3981
17 M 2802 41 OBG
MULLINIX, JANICE M. 3243 MURDOCK,67208
685-2377
47 F 3006 73 N
MURPHY, BARRY L. 3243 EAST MURDOCK #500,67208
684-0251
45 M 1902 71 IM
MURPHY, DUANE A. 3243 E MURDOCK,67208
685-1491
32 M 1902 65 ORS
MURPHY, PAUL M. 3600 E HARRY,67218
685-1111
28 M 3006 51 R
MURRAY, KENT B. 841 N BROADWAY,67214
263-6131
47 M 3901 73 IM
MYERS, AGNES E. UKSM-WICHITA,67214
688-2468
29 F 2803 63 PO
NELSON, GERALO O. 825 N EMPORIA,67214
262-3495
34 M 1902 60 PS
NELSON, RUSSELL ALAN, 3333 E CENTRAL,67208
685-5271
18 M 1902 45 PD
NELSON JR, GUST H. 3600 E HARRY,67218
685-1111
23 M 1902 46 OR
NESMITH, LESLIE W. 3333 E CENTRAL,67208
683-5611
40 M 1902 66 OPH
NEWBY, JAMES P. 905 N EMPORIA,67214
263-0296
34 M 1902 59 TS
NEWSOM, F CARTER, 3310 E DOUGLAS,67208
685-1443
18 M 1201 43 P
NIXON, WILLIAM A. 3333 E CENTRAL,67208
683-6622
16 M 1902 44 GS
NORRIS, ROBERT P. 3244 E DOUGLAS,67208
689-9232
17 M 1902 43 IM
NORTH, DORIS G. 1148 S HILLSIDE,67211
684-5257
16 F 1902 47 FP
NORTH, VICTOR, 1148 S HILLSIDE,67211
684-5257
18 M 1902 47 FP
NORTON, ROBERT K. 3244 E DOUGLAS,67208
689-9235
32 M 1001 57 PD
NOWLIN, NANCY S. 1001 N MINNEAPOLIS,67214
268-8378
F 1902 74 IM
NYBERG, FREDRIK F. 3333 E CENTRAL,67208
682-6511
22 M 2101 46 GYN
O'DONNELL JR, LEONARD A. 8033 E DOUGLAS,67207
684-2835
27 M 1902 55 IM
O'DONNELL SR, LEONARD A. 8033 E DOUGLAS,67207
684-2781
02 M S606 26 IM
OCHSNER, BRUCE B. 103S N EMPORIA STE 21S,67214
263-6273
39 M 1902 6S OPH
ODENHEIMER, BURTRAM J. 3244 E DOUGLAS,67208
689-9137
48 M 2105 73 N
OOULIO, PERLITA, ST JOSEPH MED CENTER,67218
68S-1111
39 F 74807 63 PM
OSBORNE, CONRAD C. 855 N HILLSIDE,67214
685-1381
38 M 1902 67 FP
OSIO, ANTONIO L. 1S20 S CLIFTON,67218
685-2371
41 M 26404 65 EM
OSOBA, WILLIAM G. 2525 W 13TH,67203
943-9391
25 M 2802 51 FP
OWEN, LARUE W. 303 S HILLSIDE,67211
684-7251
19 M 1902 50 ANES
OWEN, PERE A. 303 S HILLSIDE,67211
684-7251
37 M 1902 64 ANES
PADGETT, WILLIAM L. 606 STRATFORD,67206
-
24 M 1902 51 FP
PAGE, RUTH, 1051 N STRATFORD,67206
684-0255
13 F 1902 43 OO
PALMER, DAVID L. 11308 VALLEY HI DR,67209
686-7161
37 M 1902 63 PATH
PARKER, HAROLD L. 2932 BENJAMIN,67204
262-3765
32 M 1902 67 FP
PATTERSON, BRUCE W. 1S20 SOUTH CLIFTON,67218
685-2371
46 M 1902 73 EM
PAY, NORMAN T. ST FRANCIS HOSP,67214
268-5914
45 M 74802 68 NR
PENCE, CHARLES D. 3244 EAST DOUGLAS,67208
689-9175
42 M 1902 68 ORS
PENNINGTON, KATHERINE, 2113 SO BLUFF CT,67218
685-5271
16 F 1902 43 PD
PETERIE, JERRY P. 1001 N MINNEAPOLIS,67214
268-8378
48 M 1902 75 IM
PETTERSON, O'RUTH S. 5205 E 21ST,67208
682-6526
19 F 1902 44 OO
PHIPPS, JACK G. 315 N HILLSIDE,67214
686-3391
21 M 1902 53 FP
PIERRON, ROBERT L. ST FRANCIS HOSPITAL,67214
-
M
PINSKER, JACOB A. 103S N EMPORIA,67214
267-8259
06 M 1902 35 IM
POLING, TERRY L. 6155 E HARRY,67218
685-2306
36 M 1902 62 FP
POLLACK, SIMON, 7523 PLAZA LANE,67206
-
M 36 OO
POLLAND OO, STEPHEN M. 1035 N EMPORIA #185,67214
265-5291
39 M 2878 68 PM
POLLOCK, ANTHONY G A. 103S N EMPORIA STE 140,67214
265-1631
45 M 8305 71 ORS
POOLE, BERNARD T. 1035 N EMPORIA,67214
264-2806
37 M S3902 62 ORS
PORTER, GARRY L. 3243 E MURDOCK STE 400,67208
686-7351
35 M 1606 61 P
POWERS, K DEAN, 2703 E CENTRAL,67214
685-1277
23 M 1902 47 OBG

PRIETO, LUIS E, 959 N EMPORIA #203,67214
265-2613
39 M 26407 65 IM
PRUITT, JOHN C, 427 N HILLSIDE,67214
686-6608
48 M 1902 74 DTD
PULLMAN, NORMAN K, 3007 E CENTRAL,67214
686-7369
21 M 3006 45 PS
PURINTON, LEW W, 1431 S BLUFFVIEW DR,67218
685-1301
23 M 1902 48 IM
PUTNAM, LYLE B, 1035 N EMPORIA,67214
262-5037
11 M 1902 36 FP
RANDALL, GEORGE R, 5205 E 21ST,67208
684-2838
43 M 2802 69 DTD
RANDLES, MICHAEL J, 959 N EMPORIA,67214
265-0789
48 M 1902 73 IM
RAUSA JR, FRANCISCO C, 1148 SOUTH HILLSIDE,67211
683-4658
42 M 74808 66 IM
RAWCLIFFE JR, ROBERT A, 732 N TOPEKA, 67214
262-3476
29 M 3501 55 DRS
RAZEK, ZACK A, 905 NORTH EMPORIA,67214
263-0296
46 M 60501 70 COTS
REALS, WILLIAM J, ST JOSEPH MED CENTER,67218
686-9432
20 M 3006 45 PATH
REAZIN, WALTER L, 1430 HOMESTEAD,67208
685-1381
30 M 1902 58 FP
REED, A J, 1520 S CLIFTON,67218
685-2371
40 M 3901 65 EM
REED, D CRAMER, WESLEY MED CNTR/PROF DEV,67214
688-2080
15 M 2802 41 ADM
REED, DAVID D, 3333 E CENTRAL,67208
685-1291
43 M 1902 69 DR
REEVES, BRADFORD F, 3333 E CENTRAL,67208
685-1291
37 M 4812 62 R
REISMAN, MICHAEL ALAN, 3243 E MURDOCK SUITE 600,67208
683-5688
50 M 4804 75 DPH
RELIHAN, DONALD A, 3333 E CENTRAL,67208
684-5158
27 M 1902 54 DPH
REMPEL, JOHN H, 3333 E CENTRAL,67208
685-1812
38 M 3901 62 PS
RHOADS, JAMES P, 3244 E DOUGLAS,67208
689-9106
34 M 3520 60 IM
RHODEN, CURTIS H, 3243 E MURDOCK,67208
684-0252
33 M 1803 59 IM
RHODES, IVAN E, 3333 E CENTRAL,67208
685-1291
25 M 3901 49 R
RHODES, LOWELL M, 315 N HILLSIDE,67214
685-1461
25 M 1902 53 FP
RICHARDSON, STEWART F, 3420 E DOUGLAS,67208
685-2321
28 M 3005 54 P
RIEDERER, ROBERT E, 1131 S CLIFTON,67218
685-4354
16 M 1902 42 FP
RIEGER, ERNEST H, 3243 E MURDOCK,67208
682-4591
29 M 1902 56 GS
RIEPE, ROGER E, WESLEY MED CENTER,67214
688-2810
46 M 1803 74 PATH
RIDROAN, HUGH O, 434 N OLIVER,67208
682-9241
32 M 5605 57 P
ROBERTS, DANIEL K, WESLEY MED CENTER,67214
688-3185
36 M 3005 61 OBG
ROBERTS, LOUIS S, 115 S RUTAN,67218
-
95 M 2834 20 OD
ROBERTSON, JOSEPH K, 905 N EMPORIA,67214
263-0296
41 M 3901 66 GS
ROBINSON, G DONALD, 3333 E CENTRAL,67208
686-6659
28 M 1902 54 PD
ROBINSON, JOHN E, 3243 E MURDOCK STE 400,67208
686-7351
32 M 6201 50 P
ROBINSON, ROBERT H, 3244 E DOUGLAS,67208
689-9445
20 M 1902 53 ANES
ROBL, DAVID A, 1301 N WEST,67203
262-3765
48 M 1902 74 FP
RODRIGUEZTOCKER, LILIA, 1111 N ST FRANCIS,67214
265-2613
21 F 27501 49 IM
ROMALIS, BRIAN E, 3420 E DOUGLAS,67208
682-5069
39 M 6201 63 P
ROSE, SHELBY D, 3333 E CENTRAL,67208
681-2741
40 M 2012 68 PATH
ROSEN, DAVID, 1035 N EMPORIA STE 265,67207
265-3226
48 M 1902 74 PD
ROSENBERG, THOMAS F, 611 N HILLSIDE,67214
684-0501
41 M 1642 68 A
ROSS, DENNIS LEE, 1035 N EMPORIA 105,67214
263-7285
47 M 3005 73 NEP
RUSSELL, PHILIP W, 3244 E DOUGLAS,67208
689-9351
22 M 1902 44 IM
SABIN JR, GEORGE M, 334 N TOPEKA,67202
265-6601
12 M 5002 39 AOM
SADIQ, SULEMAN, 1144 N ST FRANCIS,67214
267-0159
40 M 70401 63 TS
SAEED, MOHAMMAD A, 1520 S CLIFTON,67218
685-2371
42 M 70404 66 IM
SALGADO, CARLOS R, 1144 N ST FRANCIS,67214
267-0159
33 M 13201 55 CD
SANDERS, GLORIA DEANNA, DSTEDPATHIC HOSPITAL,67212
945-9161
50 F 1902 75 PATH
SANTOSCOY, GILBERT S, 3244 E DOUGLAS,67208
689-9124
38 M 4812 62 GS
SCANLAN, TIMOTHY M, 1122 N TOPEKA,67208
265-2876
46 M 2604 71 FP
SCHILTZ, FRANCES, 115 S RUTAN #108,67218
-
93 F 6701 23 DD
SCHLACHTER, ERNEST R, 406 E CENTRAL,67202
265-0705
24 M 1902 52 FP
SCHLICHER, JOHN E, 3244 E DOUGLAS,67208
689-9346
40 M 1803 66 O
SCHLUETER, JOHN J, 3333 E CENTRAL,67208
685-9289
31 M 3841 56 R
SCHNELLE, JOACHIM, 4145 EAST KELLOGG,67218
682-0621
44 M 40933 70 FP
SCHOPF, CLIFTON C, FAMILY PRACTICE CLINIC,67214
262-3765
29 M 1902 57 FP
SCHWARTZ, V DEAN, 400 N WOODLAWN #4,67208
684-3881
24 M 1902 48 FP
SCOTT, VINCENT L, 72 MISSION RD,67207
-
03 M 3806 29 DD
SCOTT, WILLIAM H, 1431 S BLUFFVIEW STE 111,67218
685-1111
41 M 4901 65 CD
SHAFER, PRESTON J, 3333 E CENTRAL,67208
682-6511
20 M 3005 46 OBG
SHAH, MUKHTAR H, 1402 HICKORY STICK CR,67230
682-6814
40 M 70404 63 P
SHAW, JAMES W, 150 S BATTIN,67218
-
98 M 1601 27 OD
SHAW, RICHARD C, 825 N EMPORIA,67214
262-3495
35 M 1902 61 PS

SHELLITO, JOHN G, 3244 E DOUGLAS,67208
689-9124
18 M 1606 43 TS
SHIFLET, ALBERT W, 841 N BROADWAY,67214
-
06 M 3901 33 OO
SHRADER, DOYLE A, 3333 E CENTRAL,67208
682-4851
16 M 1902 41 EENT
SIEGEL, ALBERT R, 3600 E HARRY,67218
685-1111
22 M 1642 47 PM
SIEGEL, CARL D, 929 N ST FRANCIS,67214
268-5000
M
SIFFORD, R LAWRENCE, 959 N EMPORIA,67214
265-0561
25 M 1803 52 IM
SKIBBA, RICHARD M, 3244 E DOUGLAS,67208
689-9477
43 M 5606 70 GE
SLUTSKY, LAWRENCE JOEL, ST FRANCIS HOSPITAL,67214
268-5922
46 M 3501 72 DR
SMITH, ALVIN L, 929 N ST FRANCIS,67214
262-6211
28 M 5606 57 PATH
SMITH, TIMOTHY WM, 1035 N EMPORIA #185,67214
265-5291
49 M 1902 74 IM
SMITH JR, WILLARD J, 851 N HILLSIDE,67214
685-1371
32 M 1611 57 U
SNYDER, GREGG M, 3243 E MURDOCK,67208
685-2377
27 M 1803 54 NS
SOLOMON, HERMAN, 835 N HILLSIDE,67214
685-4395
37 M 2701 62 D
SOLTZ, ROBERT A, 3244 EAST DOUGLAS,67208
689-9381
47 M 2803 74 PD
SOMERS, MARVIN M, ST FRANCIS HOSP,67214
262-6211
23 M 1902 48 R
SPANN, RICHARD W, 3243 E MURDOCK,67208
684-0252
40 M 1902 65 PUD
STANLEY, KENNETH E, 959 N EMPORIA,67214
267-0256
31 M 1902 56 U
STARK, JAMES R, 3244 E DOUGLAS,67208
689-9422
20 M 1902 44 R
STECKLEY, RICHARD ALLEN, 1035 EMPORIA SUITE 130,67214
263-3271
49 M 2105 74 IM
STEIN, PAUL S, 3243 E MURDOCK,67208
685-2377
40 M 3305 66 NS
STEWART, JACK T, 4815 E CENTRAL,67208
683-8401
30 M 2802 57 IM
STOLZ, ELMER G, 425 E MURDOCK,67214
265-6287
17 M 3006 45 FP
STOSKOPF, LAWRENCE E, 1803 SIEFKIN,67208
685-4389
39 M 1902 72 ANES
STREET, DAVID E, 905 N EMPORIA,67214
263-0296
35 M 2101 61 GS
STRICKLAND, MAURICE H VAN, 3244 E DOUGLAS,67208
689-9156
51 M 4804 74 A
STRYKER, TERRY MARGARET, 3243 E MURDOCK - STE 300,67208
685-8231
47 F 1002 75 FP
SUERO, JESUS T, 1152 SOUTH CLIFTON,67218
681-1671
33 M 74802 PUD
SULLIVAN, CORNELIUS J P, 1431 BLUFFVIEW DR,67218
685-4851
18 M 3509 43 OBG
SULLIVAN, LEONARD L, 3244 E DOUGLAS,67208
689-9454
35 M 1902 61 PD
SWANSON, HOWARD J, 6611 E CENTRAL,67214
686-1152
47 M 1902 73 IM
SWEENEY, JAMES G, ST FRANCIS HOSP,67214
268-5000
M
SWISHER, WILLIAM C, 5025 E KELLOGG,67218
682-1534
21 M 1902 47 OBG
TATPATI, DANIEL A, 1144 N ST FRANCIS,67214
267-0159
44 M 49535 67 TS
TATPATI, OLGA ADELINA, U K S M - WICHITA,67214
262-8271
44 F 49535 67 PO
TAYLOR, RICHARD J, 929 N ST FRANCIS,67214
262-1952
21 M 3006 49 PATH
TAYLOR, STEVEN L, USAF HOSP,67210
681-5866
46 M 1902 77 GP
THELEN, J CHRISTINE, 212 BITTING BLDG,67202
267-3936
13 F 5104 37 OBG
THOMPSON, DANIEL M, PO BOX 4044,67204
838-3381
19 M 1902 50 FP
TIHEN, EDWARD N, 3244 E DOUGLAS,67208
689-9481
24 M 1606 48 IM
TIHEN, HENRY N, 1227 N RIVER BLVD,67203
-
96 M 1601 19 OO
TILLER, GEORGE R, 5101 E KELLOGG,67218
684-5255
41 M 1902 67 AM
TILTON, FRANK M, 3244 E DOUGLAS,67208
689-9137
33 M 2002 59 N
TINKER, ROBERT C, 1126 S CLIFTON,67218
682-7578
31 M 2834 57 ORS
TINTEROW, MAURICE M, 3333 E CENTRAL,67208
685-4389
17 M 4802 41 ANES
TIPPIN JR, ERNEST E, 959 N EMPORIA,67214
265-5256
24 M 1902 50 OTO
TOCKER, ALFRED M, 1111 N ST FRANICS,67214
265-2613
15 M 4802 40 TS
TONN, GERHART R, 855 N HILLSIDE,67214
685-1381
16 M 1902 44 FP
TRACY, TERRY A, 3333 E CENTRAL,67208
686-7301
35 M 2803 61 OBG
TREES, DONALD P, 1403 S HYDRAULIC,67211
-
14 M 4101 39 OO
TRETBAR, HARVEY A, 3243 E MURDOCK #500,67208
684-0251
25 M 1902 52 IM
TREWEEKE, MICHAEL W, 2916 EAST CENTRAL,67214
684-5234
46 M 1902 72 IM
TRUDEAU, DAVID L, ALCOHOL TREATMENT UNIT,67218
685-1111
40 M 2604 66 ADT
UHLIG, PAUL J, 3244 E DOUGLAS,67208
689-9191
28 M 1902 57 PD
VAN DER VLIET, STEPHEN K, 3244 E DOUGLAS,67208
689-9316
42 M 1606 68 OPH
VAN HOUDEN, CHAS E, 5006 LOOMAN,67220
-
M
VIN ZANT, LARRY E, 1431 BLUFFVIEW,67219
682-6532
10 M 1902 40 GS
VINE, DONALD LEE, 3244 E DOUGLAS,67208
689-9240
39 M 511 66 CO
VINZANT, WHITNEY L, 1431 SOUTH BLUFFVIEW #109,67218
686-1991
65 M 1902 71 GS
VOPAT, RICHARD L, 4805 E ENGLISH,67218
-
54 M 1902 MOST
VORHEES, VICTOR J, 3243 E MURDOCK,67208
685-8231
36 M 1902 68 FP
VOTH, DOUGLAS W, UKSM-WICHITA DEPT OF IM,67214
268-8378
34 M 1902 59 IM
WADUD, ABDUL, 1543 S HILLSIDE,67211
682-6814
35 M 70409 60 P

WALSH, RICHARD A, UKSM-WICHITA,67214
268-8224
25 M 3519 48 GS
WALTERS, ROBERT MERRILL, 3244 E DOUGLAS,67208
789-9495
41 M 2802 74 ORS
WARREN, LLOYD P, 5205 E 21ST,67208
683-7223
11 M 1902 36 OPH
WARREN, WIRT A, 352 N BROADWAY,67202
267-5031
09 M 2802 33 GER
WARREN JR, JOHN W, 3244 E DOUGLAS,67208
689-9239
15 M 2501 39 U
WEAVER, J ROBERT, 959 N EMPORIA,67214
265-5731
21 M 1902 48 FP
WEAVER, JACK O, 959 N EMPORIA,67214
265-7241
16 M 2802 42 OPH
WEBER JR, HUGO P, 1035 N EMPORIA,67214
263-7285
40 M 702 66 IM
WEBSTER, BOBBY W, 3333 E CENTRAL SUITE 303,67208
685-0559
48 M 4802 74 OBG
WEDIN, PAUL H, 1 PEACH TREE LANE,67207
685-1111
08 M 1902 38 AOM
WEIPPERT, EDWARD J, 10300 W MAPLE,67209
943-3271
44 M 1902 70 FP
WELCH, LAUREN K, 3243 E MURDOCK,67208
685-2377
35 M 1902 61 N
WELCH, MARTIN H, UKSM-WICHITA,67214
685-2221
36 M 5605 61 IM
WELLS, MAX MICHAEL, WESLEY MEDICAL CENTER,66214
688-2820
50 M 1902 76 PATH
WELLSHEAR, CHARLES C, PO BOX 8037,67214
684-0201
30 M 4705 58 P
WENINGER, JOHN H, 1148 S HILLSIDE,67211
682-6523
32 M 3005 62 FP
WEST, WILLIAM T, 3244 E DOUGLAS,67208
689-9234
24 M 1902 49 OBG
WHALLON, JACOB T, 202 N ROCK RD APT 120,67206
-
14 M 1720 41 OO
WHEELER, NICKY RAY, 3007 E CENTRAL,67214
686-7369
48 M 1902 74 PS
WHEELER, PINCKNEY R, 2208 W 13TH,67203
943-2118
18 M 3901 56 FP
WHITAKER, JAMES A, 3243 E MURDOCK,67208
684-0251
44 M 1902 72 IM
WHITE, CHARLES M, 3244 E DOUGLAS,67208
689-9422
15 M 3005 41 R
WHITE, JOHN D, ST FRANCIS HOSP,67208
268-5050
47 M 1803 73 IM
WIBERG, THOMAS A, PO BOX 594,67201
263-3271
48 M 2604 74 CO
WILDER, LOWELL W, 3333 E CENTRAL,67208
684-5158
35 M 4109 62 OPH
WILKINSON, LARRY K, 1520 S CLIFTON,67218
685-2371
46 M 1902 74 FP
WILLIAMS, CHARLES L, 3244 E DOUGLAS,67208
689-9268
16 M 2834 43 IM
WILLIAMS, HOWARD V, 1439 GOEBEL CIRCLE,67207
296-3471
18 M 1205 43 P
WINCHELL, H H FORSYTH, P O BOX 11344,67211
265-1684
34 M 3545 58 R
WINCHESTER, EUGENE B, 1148 S HILLSIDE,67211
684-0271
18 M 3006 56 FP
WISDOM, JAY K, 1148 S HILLSIDE,67211
684-0271
12 M 1902 42 FP

WISNER JR, HARRY J, 3244 E DOUGLAS,67208
689-9169
17 M 3005 43 IM
WITTMANN, ALBERT F, 2323 N WOODLAWN,67220
-
10 M 2834 38 OO
WOLF, PATRICK G, 210 S GLENN,67213
265-3979
52 M
WOLFE, FREDERICK, 6611 E CENTRAL,67206
686-1152
36 M 3508 66 RHU
WOOD, GARY B, 1148 S HILLSIDE,67211
684-2131
21 M 2802 45 IM
WOODARD, LAWRENCE E, 3244 E DOUGLAS,67208
689-9234
15 M 1605 41 OBG
WOODHOUSE, CHARLES L, 959 N EMPORIA,67214
265-8821
10 M 1902 34 ENT
WRAY, ALEXANDER J, 120 E 21ST ST,67214
838-4912
19 M 1902 49 FP
WU, JIN-TZE, 3333 E CENTRAL SUITE 214,67208
688-2920
41 M 38502 67 TR
YOUNG, DOUGLAS L, 3244 E DOUGLAS,67208
689-9213
42 M 1902 71 IM
YOUNGBERG, DEAN I, 2916 EAST CENTRAL,67214
684-5243
46 M 1902 72 IM
ZARNOW, HILARY, 8610 BRENTMOOR,67206
263-3219
45 M 1611 69 R
ZATZKIN, JAY B, 1035 N EMPORIA,67214
262-4467
46 M 2002 74 IM
ZIMMERMAN, KENNETH D, 934 CRESTLINE,67212
687-3925
29 M 3901 65 OM
ZONGKER, PHILIP E, 3244 E DOUGLAS,67208
689-9422
43 M 1902 70 R

WILSON—913
(Central Kansas Society)

OLABAL, FRANK A, ,67490
658-2400
07 M 1902 45 FP

WINCHESTER—913
(Jefferson County Society)

HUSTON, FRANCIS W, ,66097
774-2150
06 M 1601 34 FP

WINFIELD—316
(Cowley County Society)

CARRO, F AURELIO, SNYDER CLINIC,67156
221-3200
23 M 27501 49 IM
COLLINS, FRANK BUSH, SNYDER CLINIC ASSOCIATION,67156
221-3200
38 M 2701 63 TS
DAEHNKE, SIGURD S, SNYDER CLINIC,67156
221-3200
26 M 3005 60 FP
DEPOE, JOSEPH H, 1806 E 19TH,67156
221-4980
31 M 1902 57 FP
HUTCHINSON, DIRK T, 1317 WHEAT RD,67156
221-3200
48 M 3901 74 IM
KAUFMAN, LELAND R, 221 WEST 8TH,67156
221-3350
33 M 1902 61 FP
KAUL, ANAND N, SNYDER CLINIC,67156
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39 M 49530 61 IM
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221-3200
40 M 502 77 GP
MAC KILLOP JR, DANIEL, 4 FLEETWOOD DR,67156
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11 M 2407 38 OO
MILLER, FRANKLIN R, BOX 544,67156
-
02 M 2401 27 IM

NEMMERS, DAVID J, SNYDER CLINIC,67156
221-3200
34 M IB03 59 1M
PUTHENPURACKAL, GEORGE, 1211 EAST 5TH,67156
221-6100
42 M 65 1M
SAMUEL, CHANDY C, 1211 E 5TH,67156
221-6100
35 M 49527 59 GS
SHAH, ASHOK H, 1317 WHEAT RD,67156
221-3200
41 M 4954B 66 OBG
SNYDER, C JOHN, 1317 WHEAT RD,67156
221-3200
3B M 2401 64 GS
SNYDER, HOWARD E, 1317 WHEAT ROAD,67156
221-3200
03 M 4102 27 GS
WELLS, ALVIN Y, 1707 E 10TH,67156
-
97 M 1902 37 OO
WELLS, BRUCE W, 221 W 8TH ST,67156
221-3350
39 M 1902 64 IM
WHITE, R BURNLEY, 117 W 9TH,67156
221-2950
24 M 1902 52 FP
WINBLAD, JAMES N, 1211 E 5TH ST,67156
221-6100
27 M 1902 53 GS

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ATKIN, JOHN O, 1004 E MADISON,66783
625-2312
35 M 3901 61 FP

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GOODIN, WALKER D, 516 IOLEWOOD DR
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2. To encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of office holders, and the pertinent qualifications of candidates for elective office; and
3. To assist physicians and others to organize for effective political action to promote civic responsibility and integrity in government.

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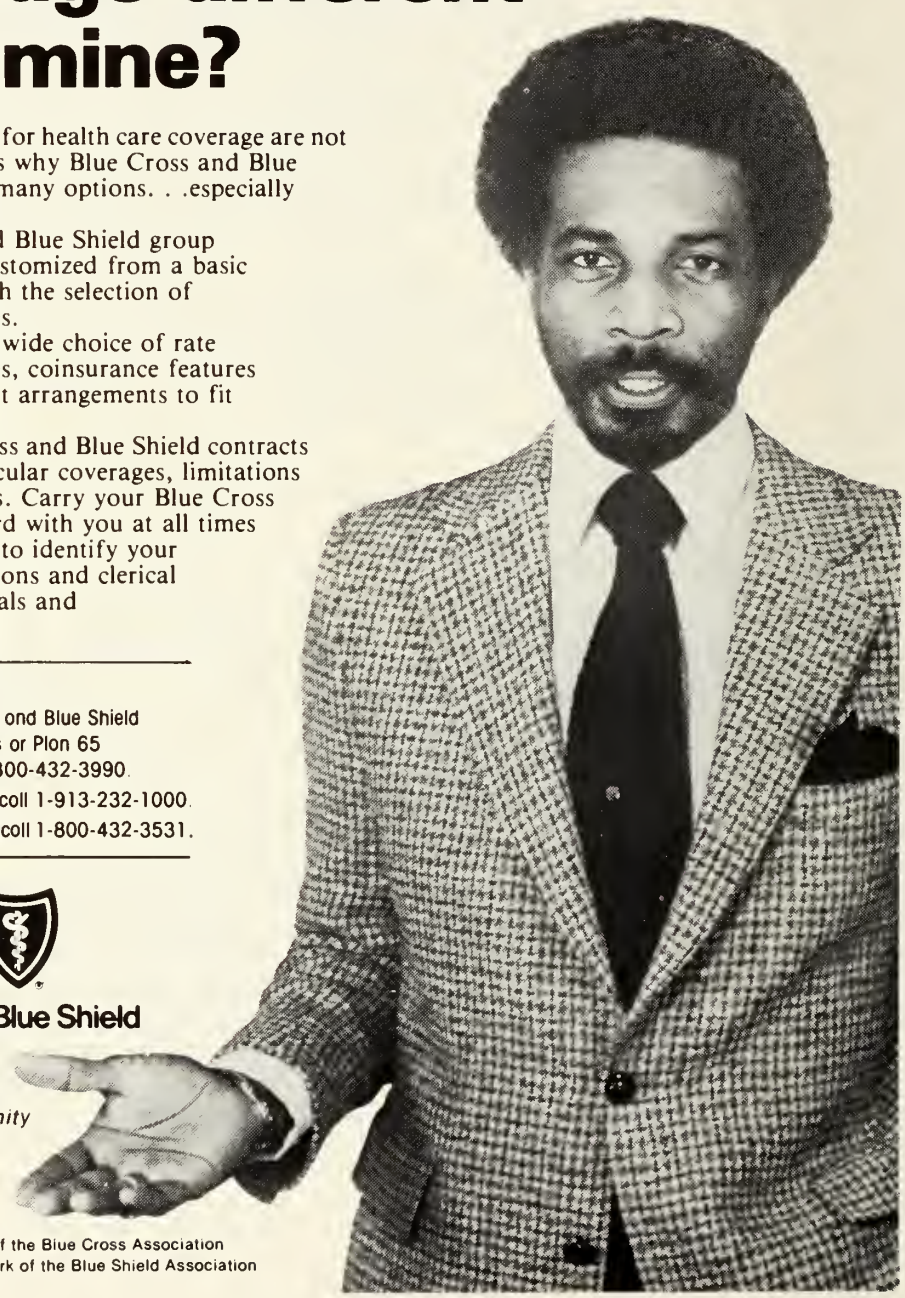
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WHEN ANXIETY AND TENSION MAGNIFY PAIN IN MUSCULOSKELETAL DISEASE*

A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC®

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.
Final classification of the less-than-effective indications requires further investigation.
The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures
Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.
USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.
Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.
Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.
PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery
Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.
ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.
A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.
Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.
More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.
Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.
Impairment of accommodation and visual acuity has been reported rarely.
OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs usually requires whole-blood transfusions.
DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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Philadelphia, Pa. 19101

FOR MODERATE PAIN

A therapeutic dose of acetaminophen in *one* tablet

A therapeutic dose of two complementary analgesics

The convenience and economy of a dosage schedule of one tablet, every four hours as needed



WHY NOT WYGESIC® ©IV

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbreviated Summary
INDICATION: For the relief of mild-to-moderate pain.
CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen
WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdoaage**)
DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine
USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and or physical abilities required for potentially hazardous tasks, e g driving a car or operating machinery Patients should be cautioned accordingly
USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards
USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group
PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol
ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.
Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions Cases of liver dysfunction have been reported
DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. (see **Warnings**) Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine
MANAGEMENT OF OVERDOSAGE: SYMPTOMS The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage end include respiratory depression (a decrease in respiratory rate and or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred
Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.
Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity A 13-gram dose has reportedly been fatal.
TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene An appropriate dose of one of these antagonists should be administered, preferably IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e g caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions
Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated Gastric lavage may be helpful Activated charcoal can absorb a significant amount of ingested propoxyphene Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed Copious gastric lavage and or induction of emesis may be indicated Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977)
Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity Serial hepatic enzyme determinations are also recommended

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage, withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. Oral—Adults: Mild and moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d., severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d. (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) Capsules, 5 mg, 10 mg and 25 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) Tablets, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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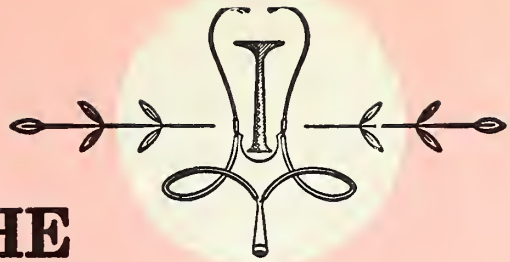
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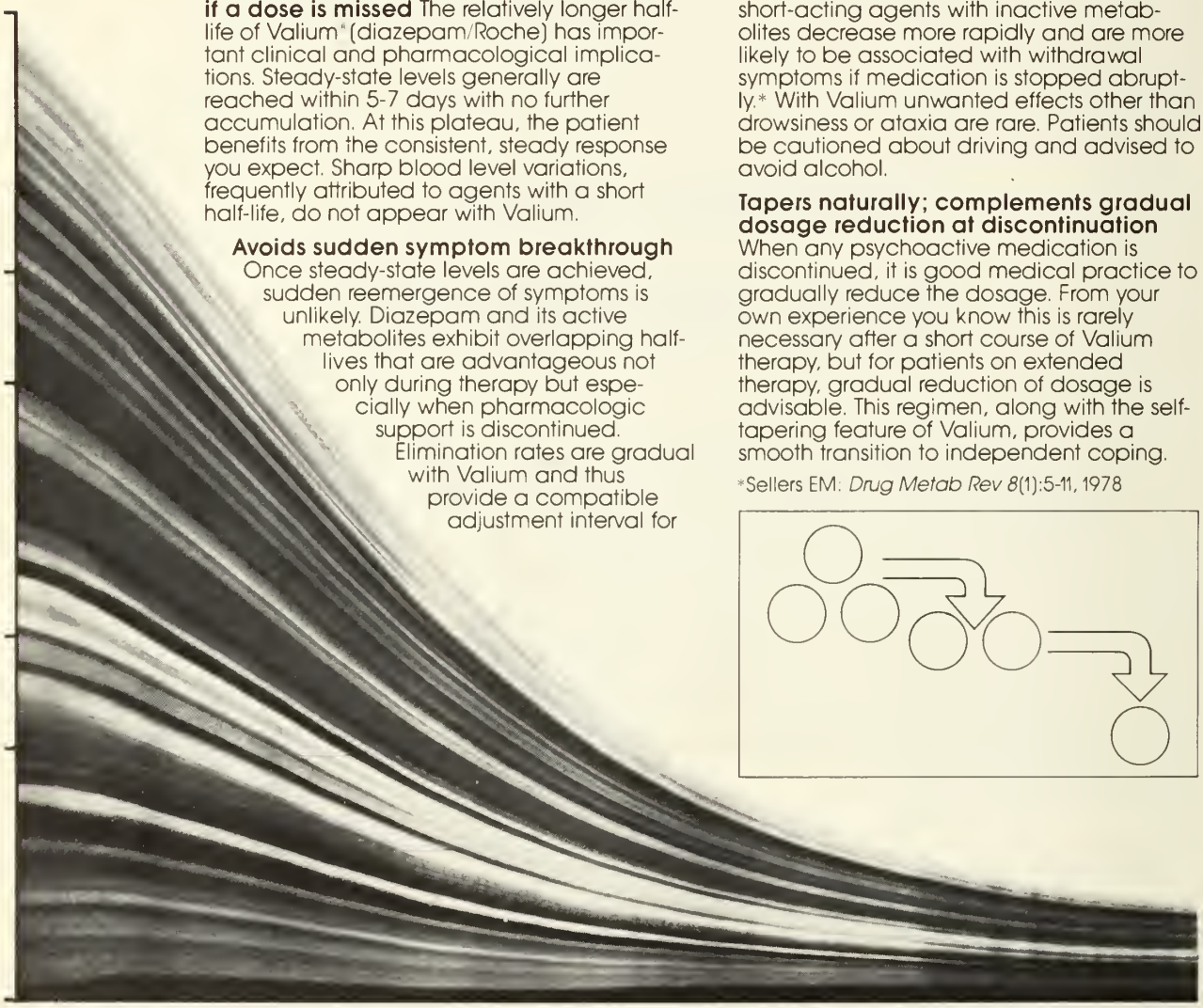
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The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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Personalities

Richard A. Walsh, Dean of the University of Kansas School of Medicine-Wichita since August 1977, resigned effective July 1. He has accepted the post of assistant area director of the Indian Health Service, a branch of the U. S. Public Health Service in Phoenix.

George Dyck, Professor and Chairman, Psychiatry, UKSM-W, has been appointed medical director of Prairie View Mental Health Center in Newton. Dr. Dyck will be director of medical education for the University at the mental health center.

Frederic Chang recently assumed responsibilities as Associate Dean for Academic Affairs at UKSM-W. Dr. Chang is responsible for curriculum development and evaluation.

Robert Manning, Professor, Internal Medicine, UKSM-W, has received the Thor M. Jager, M.D. Award for teaching excellence. Voted on by senior medical students, the award is presented to a member of the clinical faculty who has demonstrated effective organization of material, intellectual honesty, stimulation of student interest, and other teaching qualities.

Garold Minns, Chief Resident, Internal Medicine, UKSM-W, has received the Dean's Award for Clinical Teaching. It is presented to a resident physician, selected by seniors, who has exemplified distinguished service in the education of medical students.

Daniel K. Roberts, Chairman, Ob-Gyn, UKSM-W, was selected to receive the Chancellor's Award for $1,000 for Teaching Excellence.

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(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows.

"Possibly" effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported; most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

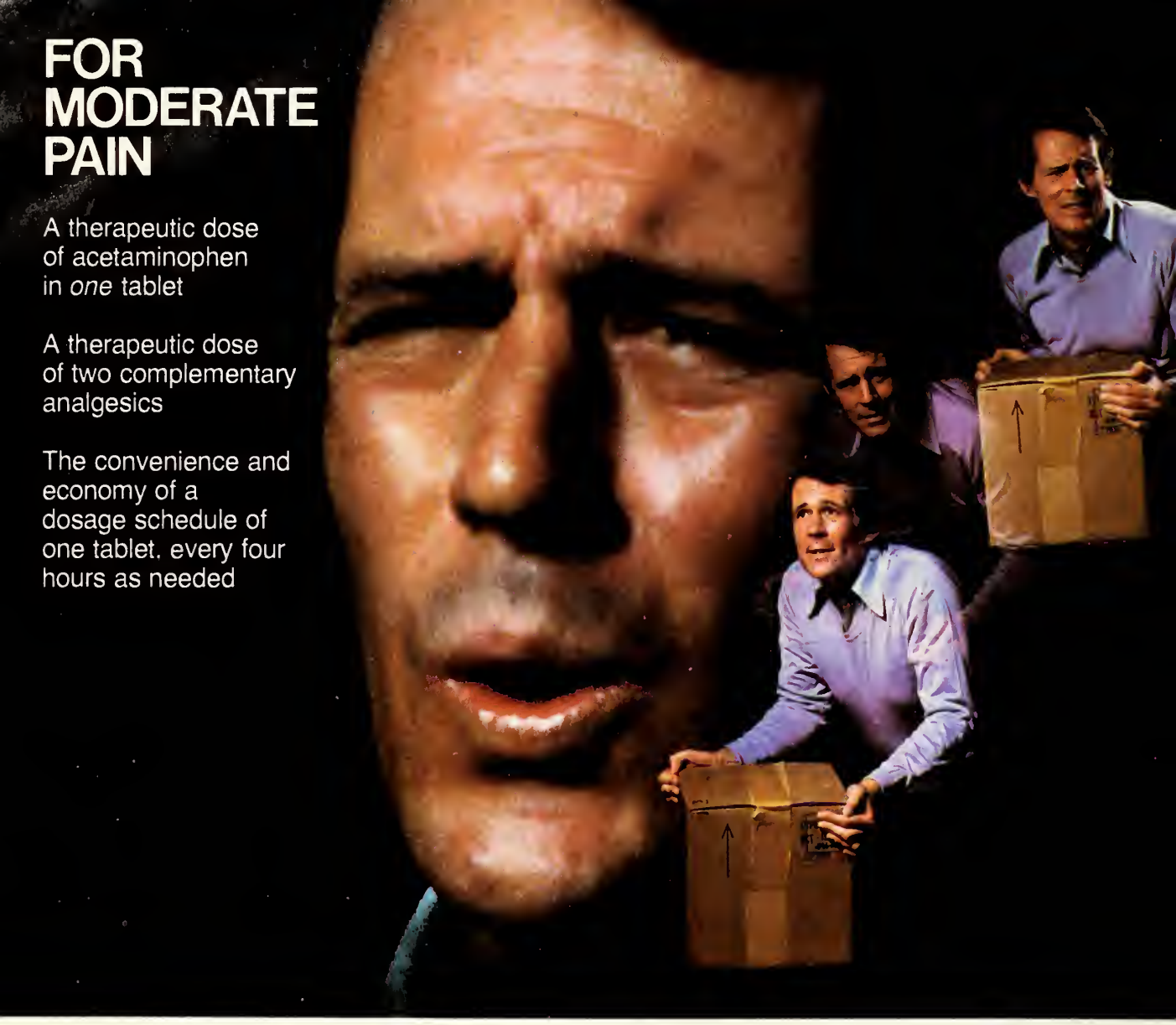
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WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**)

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery Patients should be cautioned accordingly

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin reshes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions Cases of liver dysfunction have been reported

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressents has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**) Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow Renal failure due to tubular necrosis, and myocardiopathy, have also been reported

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphen, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered, preferebly IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures Aneleptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated Gastric lavage may be helpful Activated charcoal can absorb a significant amount of ingested propoxyphene Dialysis is of little value in poisoning by propoxyphene alone Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed Copious gastric lavage and or induction of emesis may be indicated Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237 2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity Serial hepatic enzyme determinations are also recommended

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American Medical Association Auxiliary
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

The Kansas Medical Society Auxiliary was well represented at the AMA Auxiliary annual convention in Chicago in July. In keeping with the decision made last year, the reference committee structure of the AMA was initiated by the Auxiliary. Three reference committee hearings were conducted: Bylaws; Finance and Membership; and Health Issues. Kathy (Mrs. Kermit) Wedel served on the Health Issues Reference Committee. All members were allowed to address the issues during the committee hearings, avoiding time-consuming discussion on the floor.

Membership, AMAERF, Health Projects, and Legislation workshops were conducted during which delegates from all states shared program ideas. Jean (Mrs. Clair) Cavanaugh presided over the AMAERF workshop; she is now national chairperson for AMAERF. The Kansas Body Pollution Center was featured at the Health Projects workshop.

Speakers included several well known personalities. Letitia Baldrige delivered the keynote address at the opening session of the House of Delegates. She has held positions in American embassies in Paris and Rome and on President Kennedy's staff, and now has her own public relations firm. Luncheon speakers were Jessica Savitch, Washington-based NBC news correspondent who has done many special reports; and C. Carson Conrad, Executive Director, President's Council on Physical Fitness and Sports.

New national officers elected include Mrs. John Vaughan (Washington), President, and Mrs. Harry Dvorsky (California), President-elect. We are proud to announce that Lela May (Mrs. Chester) Young was elected to the 1980-81 nominating committee.

Detailed reports from each delegate who attended the convention appear in the current issue of the *KMSA Communique*. Doctor, I hope you have read the latest issue sent to your spouse and that you are anticipating future issues.

The Communique is published four times per year by the Auxiliary. The current editor is Kathy (Mrs. Lawrence) Tretbar who is Public Relations Director of Tiffany's Attic Playhouse in Kansas City and a staff writer and editorial board member of *Kansas City* magazine. Some of the pictures in the current *Communique* were taken by her husband. Larry, at the State Meeting. We will be looking forward to additional help from Larry with future issues.

Sincerely,
Evelyn Huff
President
Kansas Medical Society Auxiliary

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.: A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

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Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness *(e.g.,* operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

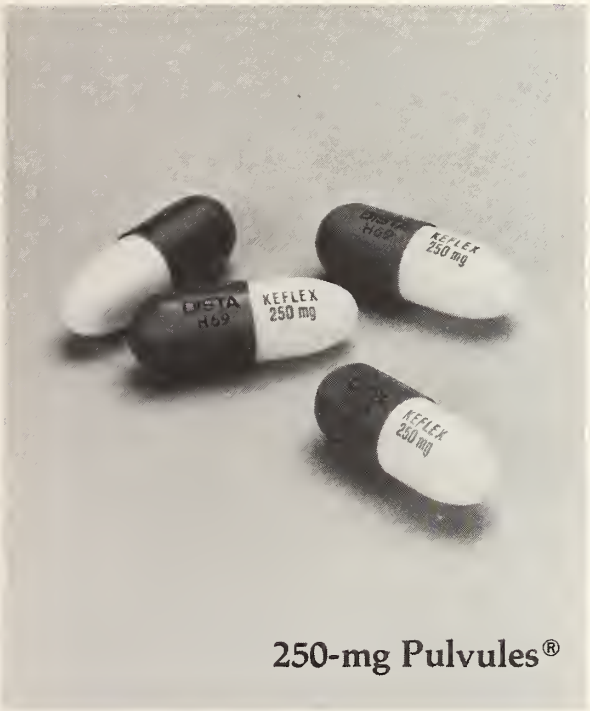
Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression: suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.,* dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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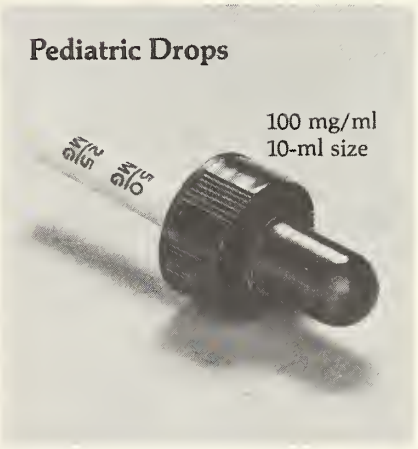
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RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
George M. Penn, M.D., Topeka (913) 272-3111 Ext. 321
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
Donald R. Tucker, M.D., Topeka (913) 354-5275
Virginia L. Tucker, M.D., Lawrence (913) 843-3750
Kermit Wedel, M.D., Minneapolis (913) 392-2144

Kansas Medical Society, Topeka (913) 235-2383/235-3619

Seventh Annual

UKSM—Wichita

Issue

The Dean's Letter—1980

The University of Kansas School of Medicine–Wichita

WILLIAM J. REALS, M.D.

The past year has been a time of refinement for the University of Kansas School of Medicine-Wichita (UKSM-W). A review of the year's activities reflects the dynamic nature of a community-based medical school as well as new stature in the Wichita community.

The installation of exit signs on Canal Route (I-135) in Wichita may not seem to be a major milestone but for UKSM-W, the signs confirmed the school has a permanent home. The facilities at 1001 N. Minneapolis on the site of the former county hospital were acquired last year and are in Phase I of the architectural renovation plan.

The year also brought administrative change. Dr. Richard Walsh resigned as dean effective July 1, 1980. An extensive search for a Family Practice Chairman ended early last year with the appointment of Dr. Edward Donatelle.

Dr. Frederic Chang was named Associate Dean for Academic Affairs and Dr. George Dyck resigned as Chairman of Psychiatry to accept a position at Prairie View Mental Health Center, although he will continue as acting chairman during the search process.

The commitment of faculty has assured continuity in medical education and expansion of clinical training opportunities. In May, 25 students graduated in the Class of '80 and in June, 49 new students arrived in the Class of '82.

In Family Medicine, UKSM-W undertook a collaborative effort with Wesley Medical Center (WMC) to direct a Family Medicine Residency Program in Wichita.

A child development program was one highlight of the year in Pediatrics. It will incorporate the Child Evaluation Center, an evaluation program in the newborn intensive care unit at WMC and a newborn follow-up program. The Kansas Regional Diabetes Center also continued to develop with the support of private funding and now serves patients in 17 states.

In Internal Medicine, research facilities were developed at the Veterans Administration Center Hospital and at UKSM-W. Fifteen residents graduated from the residency program and two of last year's chief residents joined the faculty.

It is particularly rewarding to see students graduated whose capabilities warrant eligibility for faculty appointments. A former student, Dr. William Klontz, was named director of the Psychiatry Residency Program and that program's first graduate was appointed to the faculty.

The Department of Ob-Gyn at Wesley continued participation in the perinatal project and sponsored the Second Annual Microscopic and Clinical Review in Ob-Gyn Pathology and Oncology which featured nationally-recognized speakers and drew participants from throughout the country. The Department of Surgery at St. Francis Hospital is looking forward to the recruitment of a kidney transplant surgeon and has regional HSA approval for a 10-bed Wichita Regional Burn Center.

The Liaison Committee on Graduate Medical Education granted full unqualified approval for an accredited four-year program for 14 residents in Pathology. Anesthesiology offered rotations to UKSM-W undergraduates as well as instruction for three residents, and became involved in acute and chronic pain clinics.

Twenty major postgraduate education conferences were sponsored or cosponsored as well as 93 one- to three-hour programs, for a total offering of 352 hrs AMA Category 1 Credit. A new outreach seminar series in Winfield provided postgraduate opportunities in Southcentral Kansas.

It was also a visible year for Outreach at UKSM-W. The award of a federal grant will increase efforts to screen minority and elderly populations for hypertension. Another grant will train community health nurses in hypertension control. The hypertension program began to emphasize follow-up and education, and screened a record number of hypertensives. The Mobile Continuing Education Program is now the largest hospital education consortium in the MINK (Missouri, Iowa, Nebraska, Kansas) area.

It was a busy year and as I assume the office of Dean, I look forward to the challenges and prospects of working with innovative faculty and staff. As a developing medical school, adequate financing is essential to many programs and I am seriously concerned about the pressures the Legislature may face as the competing demands for limited dollars are weighed. It may be a difficult time for a growing institution. The commitment of volunteer faculty and community resources will continue to be our strongest asset.

It is my personal goal to maintain and foster an academic environment that will provide quality education for students, research opportunities to attract and retain qualified faculty, and service to the State of Kansas as a clinical campus of the University of Kansas School of Medicine.

Departmental Reports

Family Medicine

EDWARD P. DONATELLE, M.D., *Chairman*

The Department of Family Medicine was initiated and began operation at the time of the development of the Wichita Branch of the University of Kansas School of Medicine. However, the beginning of a formalized program in graduate education for family practice began in 1969 at Wesley Medical Center; this program laid the foundation for the development of the Department long before the official initiation of UKSM-W. Although this was a community program not related to a medical teaching institution, it provided stimulus for other such programs to be developed in the Wichita area. Development of family practice residencies at St. Joseph and St. Francis Hospitals ensued, and most recently a residency in family practice was established in Salina.

The present administrative structure and faculty of the department are represented by a Department Chairman, Vice Chairman, and 9.7 equivalent full-time faculty positions. These positions represent Program Directors, Associate Directors for the two divisional University programs at Wesley Medical Center and in Salina, and community part-time faculty.

Department projections of future activities relate to all levels of medical education. The Department plans to work with the Department of Family Medicine in Kansas City to effect curricular modifications at the beginning level of undergraduage education so that the student will be prepared to meet the needs of patients through a career of primary care medicine (family practice, internal medicine, and pediatrics).

The Department will expand efforts toward increasing curricular time for family practice education during the third and fourth years from the current two months to three months.

Considerable effort will be devoted toward introduction of geriatric medicine to all students in both the undergraduate and graduate curriculums.

Cost effectiveness and cost awareness can no longer be treated as a passive annoyance by medical educators, but must be introduced as a curricular item in a structured course of medical economics early in the medical student's education.

Patient education in health care matters has become a major responsibility of medical educators. Concepts to effectively introduce this responsibility to both undergraduate students and residents must be structured, implemented, and evaluated.

Service in the Department of Family Medicine in an academic institution relates to patient care, responsibilities to other practicing physicians in the field, and to the institution itself. These services will be strengthened and continued.

Finally, research in family medicine will be actively approached as a major departmental responsibility.

Internal Medicine

DOUGLAS W. VOTH, M.D., *Chairman*

The Department of Medicine on the Wichita campus is truly citywide; thus it exemplifies the functional concept of a clincial Community School of Medicine. The vast majority of our 110 faculty members are active volunteers from Sedgwick and neighboring counties; they have responsibilities for student, resident, and continuing education at three private hospitals and the Veterans Administration Medical Center. Nine community physicians receive a modest stipend for serving as part-time faculty members. Four faculty members carry full-time responsibilities for education at the Wichita Veterans Administration Medical Center; six others have primary responsibilities at the Medical School building where the department offices are located, or at one of the private hospitals. Research projects by our faculty members have been initiated and are being conducted in the study of pulmonary diseases, hypertension, endocrinology, pharmacology, and infectious diseases.

The 12-week Obligatory Clerkship in Internal Medicine for junior students is predominantly a general medicine experience. Education for students in the sub-specialties as well as advanced general medicine is available through a series of electives offered at the hospitals and in some faculty offices; such multiple clinical opportunities are offered year round. Our faculty offers highly individualized teaching and supervision as needed for all students who select this campus for completion of under-

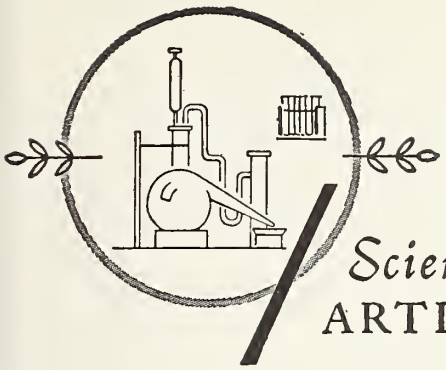
graduate medical education. Interestingly, more and more students from other medical schools are requesting clerkships offered by our Department. A detailed review of the Obligatory Clerkship in the Department of Medicine was recently published in *The Journal* (Basic Clerkship: Students in Internal Medicine on a Community Clinical Campus. *J. Kans. Med. Soc.* 80:409-413, 1979).

The program now includes 45 residents in Internal Medicine; 12 of them will complete their third year of training this year. Solomon Papper, M.D., Distinguished Professor of Medicine and Head of the Department at the Oklahoma University Health Sciences Center, was the guest speaker at our third annual graduation breakfast honoring these graduates. All of our positions are filled for the coming year; included will be chief residents (at the R4 level) at each of our integrated hospitals — St. Francis, Wesley, and Veterans Administration. The program is well established, competitive, and highly sought by interested and qualified applicants. The American Board of Internal Medicine and the Liaison Committee on Graduate Medical Education have recognized and accredited the residency program as fully integrated through our Department.

We are proud that 38 of 53 (72%) of our residents graduating in the past six years, who are not in military service or fellowship training, have been recruited to practice Internal Medicine in Kansas.

Due to the capable efforts of Internal Medicine faculty practicing and teaching at sites outside of Wichita, many of our second and third year residents are able to participate in Outreach rotations. Faculty members residing in Sedgwick County collaborate with Outreach faculty in educational programs for practicing physicians as well as for our residents and student preceptees who are there at the time.

Harmonious and cooperative relationships have been maintained with other Departments and the community at large. Thus, we are encouraged strongly to improve the attractive opportunities for our most important resources — the young men and women who choose to come to Wichita branch for their clinical training as students and residents in Internal Medicine.



Cimetidine for Trauma

Not a Panacea

STEPHEN W. MYRICK, M.D.* *and* FREDERIC C. CHANG, M.D.†, *Wichita*

A prospective study was undertaken to determine the relative effectiveness of cimetidine in prevention of UGI bleeding in trauma patients. The one-year study of severely injured patients admitted to the Trauma Unit at St. Francis Hospital (Wichita) showed cimetidine to be no more effective than nasogastric suction in prevention of UGI hemorrhage.

One complication seen in trauma patients is upper gastrointestinal (UGI) hemorrhage. This is usually in the form of stress ulcers, but other forms of peptic ulcer disease can originate these bleeding episodes.[1] Until recently treatment of this entity was directed toward stopping the already established bleeding episode, either with iced saline lavage or surgical intervention.

Since the introduction to clinical use of cimetidine in 1978 and the use of antacids for raising gastric pH, attention has been focused on the prevention of UGI bleeding in the acutely stressed patient.[2, 3] Several studies have reported conflicting data regarding the efficacy of cimetidine in prophylaxis of UGI bleeding.[4-8] These reports, in addition to several local cases of failure with cimetidine in preventing this complication, led to a prospective study using cimetidine in the acutely injured patient.

Methods

A controlled, prospective study was designed in an attempt to determine if cimetidine is more effective than standard methods in the prevention of UGI bleeding in trauma patients. From September 1978 to September 1979, severely injured patients who were admitted to the Trauma Unit at St. Francis Hospital were separated into two groups on the basis of randomly assigned hospital numbers (odd *vs.* even). All patients received standard immediate evaluation and resuscitation efforts, followed by surgery if indicated. In addition, one-half received 300 mg cimetidine intravenously as early as feasible (usually within two hours), and this dosage was continued every six hours, along with nasogastric suction, until the patient's overall condition was stable. The other half of the patients served as controls and were treated in an identical fashion except that cimetidine was omitted. Nasogastric suction was used to remove gastric acid and to monitor for bleeding. Eighty-eight patients were entered in this study during its twelve-month course *(Table I)*.

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TABLE I
TYPES OF TRAUMA

	Cimetidine	*Control*
Motor vehicle	25	25
Gunshot	7	12
Stab	2	5
Fall	4	2
Other	4	2

These patients were closely monitored in the intensive care unit (ICU) and were observed for signs of UGI bleeding. When it occurred, UGI hemorrhage was divided into two degrees:

Minor – defined as blood in nasogastric tube, usually of a transient nature and not resulting in a drop in hematocrit or requiring transfusions; and

Major – persistent bleeding of a quantity enough to be more than 500 cc in the nasogastric aspirate; to cause a drop in hemoglobin greater than 3 mg/100 ml; to require transfusion of more than two units of blood; or to cause shock.

Other parameters evaluated included age and sex, type and severity of injury, length of stay in ICU and total hospitalization, operative procedures required, number of transfusions required, mortality, side effects of cimetidine, past medical history of peptic ulcer disease, the presence of clotting abnormalities, and complications encountered during hospitalization.

Results

Of 88 patients, 46 were randomized into the control group and 42 were administered cimetidine. The types of trauma sustained by these patients are seen in Table I. The two groups were closely matched; the average age for the control group was 20.4 years and for the cimetidine group 27.6 years (range from 1-72 years). The number of males and females was evenly distributed between the groups with an overall ratio of 76:12. Nine of the cimetidine patients were in shock (blood pressure less than 90 mm Hg) upon admission; seven controls were hypotensive. The average ICU stay was 3.5 days for the cimetidine group and 4.7 days for the controls. Total hospital stay averaged 22.7 days for the cimetidine patients and 17.9 days for the controls. Those patients treated with cimetidine were given the drug for an average of 17.6 days. Of the patients who developed UGI bleeding, one patient in each group was hypotensive on admission, and only one patient (control group) had prolonged hypotension (more than 12 hours).

TABLE II
SURGICAL PROCEDURES

	Cimetidine	*Control*
Exploratory laparotomy	18	23
Thoracotomy	2	5
Tube thoracostomy	5	15
Craniotomy	7	1
Open reduction/internal fixation	4	4
Debridement in OR	4	
Application Gardner tongs or halo	3	8
Other	1	7

In the control group, 44 operative procedures were required during hospitalization, while the cimetidine group required 63 (not a statistically significant difference). These procedures are noted in *Table II*. Of the control patients, 26 (61%) required transfusion at some time during their hospital course, while 25 (76%) in the cimetidine group needed transfusion (not statistically significant). Of those needing transfusion, the control group averaged 6.3 units; the cimetidine group 8.0 units. There were five deaths (11.9% mortality) in the cimetidine group and three deaths (6.5%) in the control group (also not statistically significant). UGI bleeding was not the cause of any deaths.

In the control group, four (8.6%) developed UGI hemorrhage — two major and two minor episodes. No surgery was required for control of bleeding. In the cimetidine patients six (14.2%) bled — two major and four minor episodes. One patient required surgery for control of bleeding and was found to have a single gastric ulcer. Again, statistical significance was not noted, although the sample size was small.

Complications seen in these patients are noted in *Table III*. Infections were most common, accounting for 80.5 per cent of the complications in this series. No significant side effects of cimetidine were noted.

Discussion

The causes of UGI hemorrhage in trauma patients not sustaining esophageal, gastric, or duodenal injury are peptic ulcer disease or superficial erosions, *i.e.* stress ulcers. Lucas and Walt[1] have shown superficial gastric erosions in 100 per cent of acutely injured patients. A recent review by Silen[9] reports that approximately 5 per cent of stressed patients

TABLE III
COMPLICATIONS

	Cimetidine	*Control*
Infection		
Urinary tract	1	4
Pneumonitis	8	6
Wound	6	1
Subphrenic abscess	1	1
Peritonitis	0	1
Fat embolus	1	0
Respiratory distress syndrome	2	1
Renal failure	1	0
Disseminated intravascular coagulation	1	0

bleed massively. Of those bleeding massively enough to require surgery, a literature review by Drapanas[10] reported mortality to range from 0-46 per cent with an overall average of 24 per cent.

Numerous theories have been proposed to explain the pathophysiology of bleeding in the stressed patient. Mucosal ischemia, increased back-diffusion of H+, a breakdown of a "mucosal barrier," and an interaction of bile salts with acid and ischemia all have their proponents. A combination of several of these theories has been formulated and is shown in *Figure 1*. The overall complexity and interaction of numerous factors is apparent; H+ cannot be assumed to be the only cause for the ulcerations but is an important contributory factor. Only in the ulcers seen in patients with massive head trauma (Cushing's ulcers) has hyperacidity been shown to be the primary causative factor.

Therefore it is easy to understand why no single therapeutic approach can be successful in the prevention of ulcer formation. Cimetidine is an inhibitor of the H+ receptors of parietal cells, hence inhibiting histamine. Histamine stimulates H+ production either as the final common mediator to the parietal cells stimulating release of H+, or in conjunction with acetylcholine and gastrin in an interaction and potentiation on the parietal cell causing H+ release. Thus cimetidine diminishes H+ production by the parietal cell. The role of antacids in raising gastric pH by neutralizing H+ produces a result similar to that seen with cimetidine.

Recently attention has been directed to monitoring gastric pH to assess adequacy of therapy. This was not done in this series. Therefore a valid question in the treatment of our patients is whether the dosage of cimetidine was high enough to raise gastric pH.

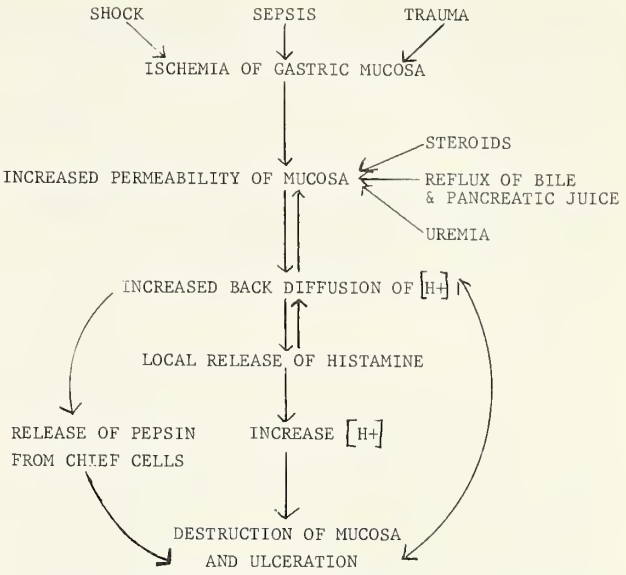


Figure 1. Pathogenesis of gastric mucosal ulceration.

Our data indicate that nasogastric suction also removes acid from the stomach; cimetidine in the recommended dosage is no more effective than nasogastric suction in preventing UGI bleeding in trauma patients. The optimal treatment of this bleeding appears to be in prevention and requires prompt reversal of shock and sepsis and vigorous treatment of any complicating factors that might lead to gastric mucosal ischemia and increased mucosal permeability. Neutralization of gastric acidity is also important and can be accomplished in a variety of ways including antacids, cimetidine, and nasogastric suction.

Conclusion

Eighty-eight patients were observed for UGI hemorrhage following trauma in a prospective randomized study. Cimetidine was no more effective in preventing this bleeding than nasogastric suction. The optimal treatment of this clinical entity lies in prevention of prolonged hypotension and prompt treatment of complications such as sepsis. A method of decreasing the gastric acidity, whether by use of cimetidine, antacids or nasogastric suction, is also needed.

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Hypertrophic Pyloric Stenosis

A Review of the Fredet-Ramstedt Pyloromyotomy

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Hypertrophic pyloric stenosis is a surgically correctable cause of progressive vomiting during the newborn period. Although this is an infrequent disorder, surgical management using the Fredet-Ramstedt pyloromyotomy represents one of the more common procedures utilized in pediatric surgery practice. The present status of incidence, cause, symptoms, physical examination, fluid management, and surgical as well as conservative therapy using ganglionparalytic drugs is reviewed in this paper.

Anatomy

THE PYLORUS, situated at the junction of the stomach and the duodenum, consists of a serosal layer, muscularis mucosa, consisting of longitudinal and circular layers, and the mucosa. Vascular landmarks include the right gastric artery superiorly, the gastroepiploic artery more inferior and anterior in the serosal layer, and the pyloric vein coursing transversely through the serosal layer at the junction of the pylorus and duodenum.

Incidence

The incidence of hypertrophic pyloric stenosis is variously reported as being between 1 : 300-1,000 live births[1, 2] in whites, and 1 : 2000 live births in black infants. Males are affected four times as frequently as females.[3]

Causes

The causes of hypertrophic pyloric stenosis remain obscure although a definite familial incidence has been noted. The lesion appears to be transmitted from a polygenetic locus with variable penetrance; an affected mother is at about four times greater risk of transmitting the disorder to her children than is an affected father.[4] Tissue examination reveals hypertrophy of the circular pyloric muscle layers and to a lesser extent the longitudinal muscle layer, edema, and a mild to moderate number of inflammatory cells. Hypertrophy of the pyloric musculature is believed to exist before birth;[4, 5] however, actual pyloric stenosis has not been observed in stillborns or premature births. The clinical observation that symptoms appear at approximately three weeks of age or later indicates that the feeding process is involved in the muscular hypertrophy and eventual stenosis. This observation led Lynn[6] to suggest that the feeding process (that is, the passage of milk curds through the narrowed pyloric channel) led to increased traumatization with resultant edema, inflammation, and further hypertrophy until stenosis occurred.

In a much quoted study, Belding and Kernohan[7] reported a decrease in the number of ganglion cells in the pyloric area. However, Friesen *et al.*[8] reported a normal number of ganglion cells in this area. This latter viewpoint seems to be the more accepted one. Friesen and Pearse[9] demonstrated a reduced histochemical reaction for esterase in the ganglion cells leading them to conclude that the ganglion cells, although present in normal number, were immature producing a hyperactivity in the pyloric area with resultant hypertrophy of the muscle layer and eventual stenosis.

Symptoms

The usual onset of clinical manifestations occurs at age 3-6 weeks. Occurrence as early as 3-5 days of life or later than 6 weeks has been reported, but is uncommon. It has been suggested that infants who are breast fed may have a later onset of manifestations. The earliest sign is regurgitation, followed by vomiting which becomes more violent in nature and in the classical case, becomes projectile, an important differential point. Vomiting in this age group is

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an ubiquitous finding. However, projectile vomiting is not. Pyloric stenosis should be considered in any child with a history of projectile vomiting.

It is important to question the mother very carefully on this point avoiding words such as "projectile" or "shooting." She should be encouraged to describe the vomiting in her own terms. Oftentimes these patients have been considered to be allergic to milk, and it is common to find a history of numerous formula changes. A decrease in the number of stools may also be observed; however, this is not a consistent finding. The presence of coffee grounds vomitus is found in less than 10 per cent of cases, secondary to the mucosal trauma of vomiting.[10] The vomiting is characteristically non-bilious since the secretions from the biliary tract do not reflux into the stomach due to the pyloric stenosis.

Physical Examination

An epigastric mass is said to be palpable in anywhere from 68-100 per cent of cases. In our experience the frequency is closer to two-thirds of the cases. Dehydration is evident on physical examination and includes the expected findings — tachycardia, decreased skin turgor, dry mucous membranes, and lack of large vein dilatation. Peristalsis may be observed in the upper quadrants and supposedly correlates to vigorous peristaltic activity in the stomach. This finding is not pathognomonic of this disease. Hypertrophic pyloric stenosis has been associated with jaundice in 2-8 per cent of reported cases and is due to an increased unconjugated bilirubin level. The cause of jaundice in this disease remains unknown. Early investigators believed that jaundice was due to extrinsic obstruction of the hepatic outflow system but recent research has shown a decrease in glucuronyl transferase activity in biopsy specimens from infants with concomitant jaundice and hypertrophic pyloric stenosis. It is felt that the combination of caloric deprivation, dehydration, and decreased glucuronyl transferase leads to the appearance of jaundice.[11]

Radiological Examination

At least one author states that upper gastrointestinal radiologic study is unnecessary in clearcut cases of hypertrophic pyloric stenosis in which the mass is palpable.[12] However, since vomiting is such a common finding in this age group, and since there is the possibility of mistaking other abdominal masses such as kidney or stool in the transverse colon for the pyloric mass, we believe that an upper gastrointestinal barium examination is a mandatory study before surgical intervention is carried out. The pathognomonic finding is described as a string sign or a railroad sign. Although normal gastric emptying of the barium contents definitively rules out the presence of pyloric stenosis, gastric retention of barium is not diagnostic since this may occur in such diseases as pylorospasm and certain diseases of the central nervous system. It has been suggested recently that abdominal sonography may aid in the diagnosis if radiographic and physical examinations are equivocal.[13]

Laboratory examination

Complete blood count studies may show the presence of volume contraction, *e.g.* increased hemoglobin and hematocrit. Electrolyte studies and blood pH usually reveal a hypochloremic hypokalemic alkalosis with a paradoxical acid urine. Hypocalcemia may accompany the hypochloremic hypokalemic alkalosis. If jaundice is present the indirect bilirubin is elevated. In these cases it is advisable to conduct clotting studies (protime and augmented partial thromboplastin time) that will measure the hepatic clotting factors (factors 2, 5, 7, 9, and 10).

Fluid Therapy

With mild dehydration, a clear liquid electrolyte solution such as Pedialyte may be sufficient for maintenance of acid-base and electrolyte status. In the more severely dehydrated child, we recommend intravenous fluid replacement with 100-120 cc/kg/24 hrs. Basal electrolyte requirements/24 hrs are: sodium, 3 mEq/kg; potassium, 2-3 mEq/kg; chloride, 3 mEq/kg; and calcium, 1 mEq/kg.

Persistent vomiting with excessive hydrogen ion, chloride, and potassium loss in the vomitus produces the hypochloremic hypokalemic alkalosis. The amount of hydrogen ion loss varies from patient to patient depending on the relative contributions of parietal and non-parietal cell secretions. This supposedly explains the range of findings in these patients from normal acid-base balance to severe dehydration and alkalosis.[13-18]

Any fluid that supplies basal electrolyte requirements and replenishes for the deficit is acceptable. We find that a fluid consisting of 5 per cent dextrose and one-half normal saline with 20-30 mEq potassium chloride/liter is a good replacement for intravenous maintenance. If there is evidence of tetany on physical examination or a decrease in serum calcium, this electrolyte should be added to the solution.

The bicarbonate content is a good measurement of the severity of alkalosis. A moderate alkalosis is

indicated by 30-50 mEq/liter, while a concentration of greater than 50 mEq/liter indicates severe alkalosis. The most immediate complication of hypochloremic hypokalemic alkalosis is cardiac arrhythmia related to the hypokalemia and alkalosis. One author[12] suggests that ammonium chloride can be added to correct severe alkalosis but we believe that this therapy is radical and rarely necessary.

The paradoxically acid urine is not a specific finding for pyloric stenosis, but is related to the hypokalemic alkalosis which produces an increase in hydrogen excretion in the distal tubule of the kidney in an effort to preserve depleting supplies of potassium. Hydrogen ion and potassium ion shifts between the intracellular and extracellular spaces may further aggravate the hypokalemia.

As the dehydration is overcome with fluid replacement and replacement of electrolytes, the hypochloremic hypokalemic alkalosis corrects. This usually takes from 24-48 hours depending upon the severity of the condition.

Operative Procedure

The operative procedure now in use was first described by Dufour-Fredet in 1907.[19] In this operation the pyloric muscle is incised longitudinally through the serosal and muscularis layers leaving the mucosal layer untouched. Fredet emphasized that closure of the muscularis layer was necessary. In 1912, Ramstedt[20] reported that closure of the muscularis layer was an unnecessary part of the procedure. This procedure remains the accepted surgical choice and is essentially unchanged from Ramstedt's description.

Operative procedure: The patient is placed on the operating table in the supine position. A towel may be rolled up and placed underneath the patient in an effort to force the pylorus anterior although this is not necessary. After proper prepping and draping of the surgical area, a transverse incision is made at the junction of the middle and upper third of an imaginary line from the umbilicus to the xyphoid on the right. A right perimedian incision may be made instead; however, we prefer the transverse incision. Using sharp and blunt dissection the incision is carried downward through the subcutaneous tissue to the anterior rectus sheath which is then incised in a longitudinal fashion. Blunt dissection is then carried down so that the rectus fibers are split longitudinally. The posterior rectus sheath and the peritoneum are opened transversely. At this point the stomach is usually visible in the incision with the gastroepiploic artery lying on its anterior surface. The stomach is then grasped and the hypertrophic portion of the pylorus is delivered through the wound. Using a scalpel, the serosal layer is then incised longitudinally. A Benson pyloromyotomy spreader is then carefully inserted in the hypertrophic muscular layer and this layer is then spread using blunt dissection downward until the mucosal layer is visible. If the mucosal layer is violated, it is important at this point to close it with a stitch. This, however, is a rare complication. The integrity of the mucosal layer may be checked by using finger pressure on the stomach to observe the passage of air and fluid from this structure through the pyloric channel. If there are any defects in the mucosal layer, air and fluid will rush out at this point. The pylorus is then replaced inside the wound beneath the liver margin which serves to protect the wound from any increased abdominal pressure. Next, we close the peritoneum and the posterior rectus sheath with 4-0 Vicryl interrupted sutures; reapproximation of the rectus muscle with 4-0 Vicryl interrupted sutures is optional. The anterior rectus sheath is then closed using 4-0 interrupted sutures, the subcutaneous tissue with 4-0 interrupted catgut sutures, and the skin with a 4-0 nylon running subcuticular stitch. The entire procedure normally takes 30 minutes or less.

Postoperative Complications

Complications — including wound infection and pneumonitis or pneumonia — are those found following any pediatric surgical procedure. Wound infection is a very rare complication if proper technique is utilized throughout the procedure. Pneumonia or pneumonitis is a rare complication with proper anesthetic management. The appearance of a shock-like condition following the surgical procedure may indicate perforation of the mucosal layer,[21] an extremely rare complication in the hands of competent pediatric surgeons. The mortality is less than 0.5 per cent with the greatest risk in patients with unconnected fluid and electrolyte imbalance. When the procedure is carried out on the patient who is in acceptable acid-base and electrolyte balance, the risk is minimal.

Postoperative Feeding

Patients are kept without oral feedings for a minimum of six hours postoperatively since it has been demonstrated that gastric emptying occurs up to six hours following the procedure, due to decreased gastric motility and ineffective emptying of the stomach contents. Our feeding regimen utilizes 10 per cent glucose-water alternated with half-strength formula. Initially the child is fed 10 cc of glucose-

water. If this is tolerated, 10 cc of half strength formula is offered two hours later. This is increased by 10 cc every two hours alternating with the glucose-water and half strength formula. The end point in the therapy is achieved when two ounces of formula are taken and retained in one feeding. Next, the child is advanced to full strength formula. If vomiting or regurgitation occurs, the patient is dropped back one step in this regimen. (For example, if a child regurgitates on 20 cc of half strength formula, two hours later 20 cc of 10 per cent glucose-water is given. If this is tolerated, the regimen continues from this point). For mothers who are breast feeding the child, the regimen starts with one minute breast feeding on one breast. Two hours later, one minute is allowed on each breast. Two hours after this, two minutes are allowed on one breast and one minute on the other breast. This continues in a similar fashion until five minutes are reached. Then, the child is fed as tolerated. If the child regurgitates on this regimen, a similar step back to the previous two-hour amount occurs and the regimen continues from this point. Intravenous fluids are continued as needed for maintenance, decreasing the amount as the oral intake is increased.

Medical Therapy

Medical therapy of pyloric stenosis is popular in Europe[22, 23] and involves the use of Atropine and related drugs (scopolamine and barbiturates). Although successful in a certain number of cases, failure rates of up to 50 per cent have been reported. Also, therapy must be continued on an inhospital basis for up to five weeks before the final results are known. Since the Fredet-Ramstedt pyloromyotomy is proven as an effective, safe, and simple procedure in experienced hands, medical therapy has been almost universally discouraged in the United States.[4, 10, 12, 24]

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Lithium Intoxication

Case Report and Review of Treatment

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THE LIFETIME incidence of major affective disorder in the population may be as high as 18 per cent.[1] The portion of that group using lithium is increasing, and primary physicians will necessarily encounter more patients taking lithium carbonate. A recent case illustrates the importance of awareness by primary physicians of the symptoms, signs, and management of lithium intoxication.

Case Report

A 52-year-old white female suffered for many years from recurrent bouts of depression manifested primarily by low mood, difficulty in concentrating, social withdrawal, and low energy levels. After observing that treatment with tricyclic antidepressants provoked hypo-manic symptoms, her psychiatrist initiated a trial of lithium carbonate. On this regimen, the patient experienced remission for 2½ years. (Dosage was 1200 mg lithium carbonate/day, and serum lithium levels averaged 0.8 mEq/1.)

Six weeks prior to the hospitalization here reported, depressive symptoms recurred. Without consulting a physician, she increased her dosage of lithium carbonate to 1800 mg/day. After four days on that dosage, serum lithium level was 0.9 mEq/1. There were no toxic symptoms during three weeks at a daily dose of 1800 mg.

Three weeks following this increase, she experienced a sudden attack of abdominal pain. Acute cholecystitis-cholelithiasis was diagnosed, and a cholecystectomy was performed. The surgeon was not aware that the dosage of lithium had been increased by the patient. Lithium carbonate, 1800 mg/day, was reinstituted as soon as oral intake was possible. She recovered uneventfully from the cholecystectomy, but experienced an episode of distal obstruction of the bile duct due to stones. She was discharged with a T-tube in place and still taking 1800 mg lithium carbonate/day. Twelve days after discharge, she began to experience tremors and — according to family reports — confusion. She complained to her primary physician of tremors, without informing him of her increased lithium intake. The physician prescribed chlordiazepoxide. Blood drawn for a routine (not stat) lithium level later that day showed serum level at 2.9 mEq/1, but the results were not available for 24 hours. During the remainder of that day, she became progressively more weak, tremulous, and disoriented. She would neither eat nor drink, nor would she consent to be fed. She was restless, sleepless, and pulled out her T-tube, which was reinserted. She was admitted for observation at which time (24 hours after the blood test showing serum lithium at 2.9 mEq/1) serum lithium measured 5.4 mEq/1. She was comatose and responded only to painful stimuli. Blood pressure was 90/60. She was mute, could not follow simple commands, had tremors and hyperactive deep tendon reflexes in all extremities, sustained clonus in wrists and ankles bilaterally, and extensor plantar responses on the right side. Urinary output was 85 ml/3 hrs. The remainder of the laboratory data on admission were as follows: hematocrit, 48.3; WBC, 22,200 with 74% leucocytes, 10% stabs; urine sp gr, 1.013 with trace proteinuria, 20 WBC/hpf and many hyaline casts; alkaline phosphatase, 366; SGOT, 46; SGPT, 208; BUN, 49; sodium, 127; potassium, 4.9; chloride, 9.6; and carbon dioxide, 25. Other admission laboratory results were unremarkable.

There is increasing utilization of lithium carbonate for treatment of affective disorders. Because of the increased risk of lithium intoxication — particularly in cases of intercurrent illness or surgery — it is essential that primary care physicians be aware of the symptoms and management of this potentially fatal condition.

The patient was treated with intravenous fluids and furosemide, 40 mg intravenously twice daily. Urine output maintained a fluctuating course despite the daily administration of furosemide *(Table I)*.

On the third day, though still somnolent, she could be aroused by strong auditory stimuli, and

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could speak a one-word answer. She exhibited an expressive aphasia, and continued to experience hyperactive deep tendon reflexes, clonus, and extensor plantar responses. Lumbar puncture revealed clear fluid under normal pressure. Cerebrospinal fluid protein was 55 and glucose 75, with no cells.

On the seventh hospital day, convulsive activity was noted. This consisted of several brief episodes — each less than one minute in duration — of lack of responsiveness, tonic clension of the jaws, and rigidity of the neck. There were no clonic movements. Electroencephalogram revealed diffuse slowing in the theta range and spike discharges throughout. Computed tomography scan of the brain with contrast revealed results within normal limits. With continued furosemide and intravenous fluids, the serum lithium slowly decreased *(Table I)*. Transient hyponatremia and hypokalemia were corrected.

On the ninth hospital day, tremors and clonus were gone, and the patient was alert and rational. Dysarthria and ataxia were still present.

On the 11th hospital day, 24-hour creatinine clearance was 21 ml/minute. After an eight-hour fast, urine osmolality was 238 mosm/kg (normal 270-900), and serum osmolality was 280 mosm/kg (normal 275-300).

On the 15th hospital day, the patient was discharged. All neurologic signs had disappeared, although the patient still complained of difficulty in concentrating.

On follow-up nine months after discharge, she remained free of neurologic or psychiatric symptoms. Repeat 24-hour creatinine clearance was 67.5 ml/minute. Urine osmolality was 431 mosm/kg, and serum osmolality was 286 mosm/kg following 12-hour fast. (Normal values in this laboratory are 500-800 and 280-295, respectively.)

Discussion

This case presents several features of importance to the primary physician.

Lithium intoxication may be precipitated by intercurrent illness, especially febrile ones. In addition to possible dehydration, a well-regulated patient may respond to intercurrent illness with decreased lithium clearance. If the accustomed dose is continued, lithium toxicity may occur. This phenomenon, first noted 20 years ago, has not been stressed sufficiently.

The most common presenting symptoms of lithium intoxication are weakness, tremors, and delirium. Convulsions and changes in the urinary output (polyuria or oliguria) may be seen.

The organs most seriously affected by lithium intoxication are brain, kidney, and heart. Common effects on the brain include tremors and delirium — disorientation, memory loss, incoherence, or a combination of these. If the intoxication is severe, coma may occur. (These signs may disappear in reverse order, *i.e.*, as coma improves, delirium may emerge.) Convulsive activity is common; it is not clear whether convulsions are a toxic or a withdrawal symptom. Clonus, positive Hoffman and Babinski signs, ataxia, aphasia, and slurred speech are also

TABLE I
CLINICAL COURSE AND PROGRESSION OF LABORATORY FINDINGS BY HOSPITAL DAY.

Hospital day		*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
Urine output (ml/24 hr)		85	1392	1635	2315	1000	1100	350	320	700	3805	2546	1775	2375
BUN (mg/100 ml)		45	29	9	10	17	28	—	68	96	96	58	58	—
Serum Li (mEq/l)	2.9	5.4 / 4.2	3.4	2.3	1.7	1.7	1.6	1.4	1.3	1.2	1.0	0.7	0.5	
Mental status		Comatose		Arousable, gives one word answers.				More alert but memory and orientation impaired.		Alert and rational				
Neurologic signs		↑ DTRs, clonus extensor plantar resp. spontaneous tremors.		Dysarthria, expressive aphasia, tremors, clonus, ↑ DTRs.				"Convulsive" Activity		Tremors, clonus, ↑ DTRs gone. Dysarthria and ataxia persist.				

common. Slurred speech and ataxia may remain as permanent sequelae.[2, 3]

Polyuria may be seen during lithium treatment, even at usual clinical dosages, and is thought to result from the reversible inhibition of adenyl cyclase in renal tubules rendering them unresponsive to antidiuretic hormone. Less commonly stressed is the fact that in significant intoxications, lithium may produce oliguria or even anuria.[4] Obviously, in such cases the ability to excrete lithium is nil and hemodialysis is the only effective remedy. Oliguria is due to a direct toxic effect on renal tubules and is reversible if the patient survives. Renal impairment manifested by decreased creatinine clearance, decreased urine concentrating ability, or abnormalities of renal tubular cells on biopsy may be a permanent sequelae of even mild lithium intoxication.

Cardiac manifestations of lithium intoxication are primarily inversion or flattening of T-waves. Occasionally hypotension and peripheral circulatory failure are seen.[4, 5]

To prevent intoxication, lithium should be utilized only when indicated for:

1. Treatment of manic states;
2. Prophylaxis in patients who suffer frequent affective episodes or mood swings;
3. Prophylaxis in patients with recurrent depression (unipolar affective disorder); or
4. To prevent recurrent rage or irritability (in institutionalized, retarded children, for example) — an application currently in the experimental stage.

Even if a patient has a disorder that might respond to lithium, the drug should still be withheld unless the physician is satisfied that renal function is adequate (by 24-hour creatinine clearance) and that the patient is reliable in following instructions. Once the decision has been made to treat with lithium, serum levels should be carefully monitored and an oral dosage chosen to maintain the serum lithium in therapeutic range (1.0-1.5 mEq/l) or prophylactic range (0.5-1.0 mEq/l) as the situation requires. Recent evidence suggests that the lower of the above ranges may be more desirable.

A patient on lithium who suffers intercurrent illness — especially a febrile one — or who undergoes a surgical procedure, should have lithium temporarily discontinued if possible. If clinical considerations require lithium maintenance during an intercurrent illness, more frequent serum lithium determinations are required. This case illustrates problems that can occur when patients and physicians do not communicate. The patient did not inform either her primary physician or surgeon of her increased lithium dosage. In some cases, to conceal a psychiatric illness, patients may not tell physicians about being on lithium at all. History taking, familiarity with symptoms of lithium intoxication, and a reasonable index of suspicion are invaluable in such cases.

Lithium intoxication is potentially fatal. Deaths are common at serum levels of 4-6 mEq/l. Permanent neurologic and renal sequelae may occur following intoxication at lower serum levels. In a patient known to be taking lithium, coarse or severe tremors, muscular weakness, disorientation, or coma should be considered presumptive signs of intoxication and indication for a *stat* serum lithium determination. (Fine, mild tremor may be seen at therapeutic blood levels and does not suggest toxicity.)

It is essential to treatment that lithium intake be discontinued and excretion enhanced, while vital functions are maintained. So long as urine output is adequate, lithium will be excreted. Therefore, maintenance of fluid and sodium intake are crucial. Urine output must be monitored. Tubular-acting diuretics may be used to increase urine output, but they do not increase lithium clearance. Aminophylline, 500 mg intravenously push or 25 per cent mannitol, 100 ml intravenously push or drip has been shown to increase renal clearance of lithium.[6] Alkalinization of urine also increases clearance. If oliguria occurs, hemodialysis will be necessary.

As in this case, the serum lithium level may continue to increase long after the last dose of lithium has been taken. This may be due to continued absorption of lithium from the gastrointestinal tract; for this reason some authors recommend gastric lavage in all patients with intact gag reflex.[7] However, the increase in serum lithium may result from shifts of lithium among body compartments, a phenomenon that probably explains the "rebound" of the serum lithium often observed after hemodialysis is discontinued.

It is important to remember that the fall in intracellular lithium follows the fall in serum lithium by a period of hours to days; therefore, neurological improvement may not be immediate. Ataxia and dysarthria may be permanent.

An interesting feature of the present case is that with conservative management — *i.e.*, without mannitol, aminophylline or dialysis — the outcome was better than often reported for intoxications in the range of 5 mEq/l treated by more vigorous methods. The significance of this observation is not yet clear.

Summary

Five guidelines for safe lithium maintenance are:

1. Use lithium only when indicated.

2. Select reliable patients. Screen them for renal impairment by means of serum creatinine or 24-hour creatinine clearance.

3. Assess serum lithium every three months, 24-hour urinary output and serum creatinine every 12 months, 24-hour creatinine clearance every two years. Thyroid status should also be assessed annually.

4. If the patient suffers intercurrent illness, discontinue lithium temporarily, if possible, or follow serum levels closely.

5. If coarse or severe tremors or impaired mental functioning occur, check serum lithium immediately.

Five guidelines for the treatment of lithium toxicity are:

1. Discontinue lithium intake. Consider gastric lavage if gag reflex is normal.

2. Monitor urine output; if patient is oliguric, arrange for hemodialysis.

3. Maintain adequate fluid and sodium intake.

4. Aminophylline, 500 mg intravenously or 25 per cent mannitol, 100 ml intravenously may be used to increase lithium clearance.

5. Prevent and detect complications such as arrhythmia, oliguria, or pneumonia by monitoring vital signs, intake and output, electrocardiogram, and chest x-rays. Serial lithium, white blood count, BUN, and creatinine will help in early detection of complications.

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Cimetidine for Trauma

(Continued from page 385)

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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The "Skinny" Needle

Percutaneous Transhepatic Cholangiography in a Community Hospital

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Percutaneous transhepatic cholangiography was originally performed in the 1930s utilizing an 18 gauge metal needle. In 1974 Okuda described a series of percutaneous transhepatic cholangiographies utilizing a 23 gauge "skinny" needle.[1] Described here are the results of percutaneous transhepatic cholangiography with the "skinny" needle in a community hospital. The procedure is simple, safe, and effective in differentiating obstructive from nonobstructive jaundice.

Methods

Nine patients undergoing ten percutaneous transhepatic cholangiographies were evaluated. Eight patients showed the characteristic biochemical and clinical signs of "cholestatic" jaundice.[2-4] One patient had suspected biliary colic *(Table I)*.

The only absolute contraindications to this procedure are bleeding diathesis, known allergy to iodinated dyes, and ascites.

All patients were without food intake for 8-12 hours prior to the procedure. Intravenous ampicillin, 500 mg, was given one hour prior to the procedure; if obstruction was found, this was continued every six hours until surgical relief of the obstruction. Under fluoroscopic guidance, a 22 or 23 gauge needle was introduced at the right midaxillary line, in a manner similar to percutaneous liver biopsy, and was aimed at the xiphoid process. Aspiration was not possible with the "skinny" needle. With the needle introduced into the liver just short of the vertebral column, a small amount of radiopaque dye was injected. The next step of the procedure was then determined by the pattern of the contrast. Three dye patterns were found to develop on fluroscopy: (1) A nodule of dye at the end of the needle suggesting that an intraparenchymal injection had been made; (2) Rapid disappearance of the dye from the area of fluroscopic viewing, indicating that the needle was in a large vessel; or (3) Bile ducts were outlined *(Figure 1)*. If either of the first two patterns was noted, the needle was slowly withdrawn a short distance and another injection was made. Once a biliary radicle was entered, Renografin, 50-75 cc, was injected to outline the hepatic and common bile ducts. All patients with biliary tract obstruction underwent surgery within 24 hours.

Use of the 22 or 23 gauge "skinny" needle for performance of percutaneous transhepatic cholangiography is a recently developed technique. This review demonstrates its feasibility, safety, and effectiveness in a community hospital setting.

Results

Table I outlines the clinical problem, liver profile, results of percutaneous transhepatic cholangiography, and sonography in the nine patients studied. The liver profile shows the typical mild elevation of SGOT and SGPT with marked elevation of the alkaline phosphatase. Patient One was an exception in that SGOT and SGPT were moderately elevated with biliary obstruction.

The biliary tree was outlined in all patients whether or not obstructed. E. coli bacteremia was the only complication. The pathology subsequently determined in these cases is listed in *Table II*.

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TABLE I

Patient No.	*Age*	*Problem*	*Bilirubin (T/D)*	*Alk. Phos.*	*GOT/ GPT*
1.	79	Jaundice	5.3/3.5	295	451/352
2.	59	Jaundice	12.3/8.4	350	75/90
3.	39	Pain	.6/.2	180	24/93
4.	68	Jaundice	4.8/3.3	1215	99/70
5.	78	Jaundice	12.3/11.4	639	50/90
6.	25	Jaundice	11.3/6.3	352	73/101
7.	60	Jaundice	6.2/3.7	705	189/295
8.	81	Jaundice	14.1/8.5	104	29/10
9.	82	Jaundice	4.0/1.4	215	54/40
10.	22	Jaundice	8.0/6.0	300	120/90

TABLE II

Pathology	*No. of Patients*	*Per Cent*
Stones	5	50
Stricture	2	20
Carcinoma	2	20
Other	1	10

In the five cases of obstruction where both sonography and percutaneous transhepatic cholangiography were performed, dilated ducts were demonstrated in all cases by percutaneous transhepatic cholangiography, but in only four cases by sonography.

A typical stricture is illustrated in *Figure 2* and obstructing calculi in *Figure 3*.

Discussion

Cholestatic jaundice can be a perplexing clinical problem because the characteristic laboratory abnormalities of biliary tract obstruction can be seen in primary hepatocellular disorders as well. This study demonstrates the use of an old technique in outlining the biliary tree using a relatively new device — the "skinny" needle.

Percutaneous transhepatic cholangiography is re-



Figure 1. A normal percutaneous transhepatic cholangiogram.

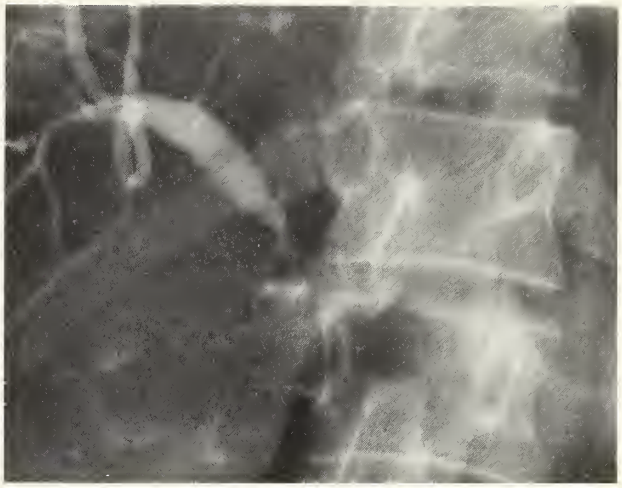


Figure 2. A typical benign stricture with a dilated common bile duct.



Figure 3. Obstructing calculi in the common bile duct.

liable in outlining bile ducts. The biliary tree was outlined in all cases attempted whether the ducts were dilated or nondilated. These findings compare favorably with other reported series of percutaneous transhepatic cholangiography.[1, 5-7] The success rate compared to that of sonography in distinguishing dilated from nondilated ducts is of note. Dilated ducts were identified in five cases. When compared to sonography in these same cases, the sonogram was positive in only four. Perhaps more significantly, percutaneous transhepatic cholangiography can identify the level of obstruction which is not as easily done with sonography.[8]

Outlining the biliary tree using the "skinny" needle proved to be safe with only one complication in ten studies. This complication, gram negative sepsis, was readily controlled with appropriate antibiotics. This rate of complication also compared favorably with other studies.[1, 5-7]

The procedure is simple, requiring expertise similar to that for safe percutaneous needle biopsy of the liver. One to five insertions were required to outline the ducts fully. A radiologist or gastroenterologist experienced in interpretation of biliary tract radiographs is an essential element in the final interpretation.

This series of ten cases of percutaneous transhepatic cholangiography supports other reports that this procedure is safe, simple, and reliable in differentiating obstructive from nonobstructive jaundice. Also, the area of obstruction and often the causative factor is identified. As demonstrated, this procedure can be utilized in a community hospital.

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CCAML

Congenital Cystic Adenomatoid Malformation of the Lung

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Congenital cystic adenomatoid malformation of the lung (CCAML), first described by Stork in 1897,[1] has subsequently been reported in more than 140 cases in English literature. Reported here are two cases of CCAML with a brief review of causes, physical findings, pathology, and treatment.

Two cases of congenital cystic adenomatoid malformation of the lung (CCAML) were diagnosed and treated at Wesley Medical Center during the last five years.

Case One. A Caucasian male infant was delivered at 31 weeks gestation by Caesarean section because of abruptio placentae. The mother, a 23-year-old gravida 2, para 1, abortus 0, was free of any prenatal problems. Immediately following delivery, the infant had spontaneous but weak respirations and required resuscitation with oxygen. Apgar score at one minute was 5. Physical examination revealed decreased muscle tone and activity with absent Moro reflex. Respirations were 40/minute with grunting, and substernal and intercostal retractions with decreased air entry. The initial report of the chest roentgenogram was reported as suggestive of early changes of hyaline membrane disease *(Figure 1)*. A barium enema ruled out a diaphragmatic hernia.

The infant's respiratory distress worsened; endotracheal intubation was performed for mechanical ventilation. The pediatric surgical consultant recommended left thoracotomy for cystic lung disease, which was performed at 2 days of age. At operation a firm, rubbery, beefy-red multicystic mass measuring 6 x 4 x 3 cm was found replacing the left lower lobe and lingula *(Figure 2)*. A resection of the left lower lobe *(Figures 3 and 4)* microscopically revealed cystic adenomatoid malformation. The infant's condition initially improved with subsequent increasing respiratory distress and he died on the day of surgery. The only significant abnormality found at autopsy was congenital hypoplasia of the right lung.

Case Two. A white male, 33 weeks gestational age, was delivered vaginally after premature labor with polyhydramnios. His mother was an apparently healthy 27-year-old, gravida 5, para 2, abortus 2 with no antepartum care. The neonate's Apgars were 6 at one minute and 5 at five minutes. Physical examination showed him to be cyanotic with poor tone; breath sounds were decreased on the left side; heart examination elicited sounds best at the right sternal border with no detectable murmur; and a left testicle was undescended. Chest x-ray showed a cystic mass in the left lung with mediastinal shift.

Pediatric surgical consultation was obtained and the x-ray findings were felt to be consistent with CCAML. Heart catheterization, performed prior to surgery, revealed dextroposition of the heart and abnormal position of the left pulmonary artery secondary to the cystic mass in the lung.

Thirty-six hours after delivery, because of continuing respiratory embarrassment, a left thoracotomy and left lower lobectomy were performed. At operation a spongy, beefy red mass 7.5 cm in greatest diameter was removed which proved to be CCAML.

Four days postoperatively he was able to breathe room air. On the 15th day he developed pneumonia due to *Pseudomonas aeruginosa;* this responded well to antibiotic therapy. He was discharged in good condition at 27 days of age.

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Figure 1. Chest roentgenogram demonstrates diffuse opacification of the entire left lower lobe. Note patchy aeration of the central portion.

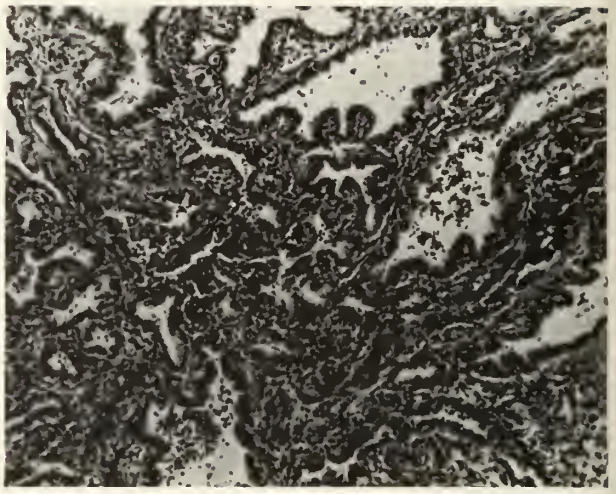


Figure 3. Picture with mucous glands; congenital cystic adenomatoid malformation. Note the multiple, irregular spaces lined by low cuboidal epithelium. Mucous glands are present in the center.



Figure 2. Picture of the specimen that included the left lower lobe and lingula.

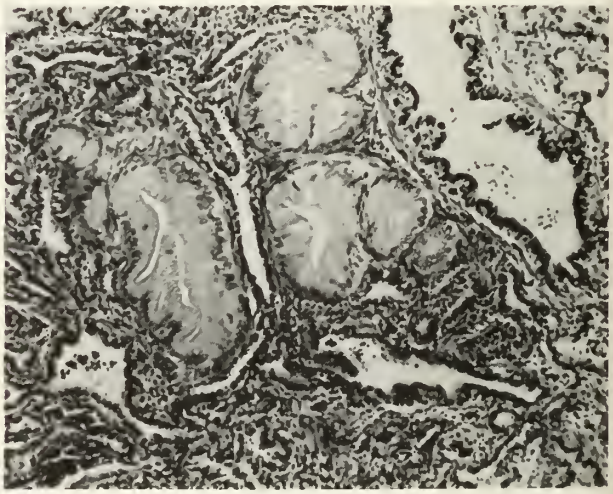


Figure 4. Picture with mucous glands; congenital cystic adenomatoid malformation. Multiple irregular spaces are lined by low cuboidal epithelium.

Discussion

Bartholin first described the broad category of Congenital Cystic Lung Disease in 1687 and the subject was introduced in the American literature in 1925 by Koontz[2]. On the basis of specific pathologic microscopic findings, Buntain[3] in 1974 defined four distinct categories previously grouped as congenital cystic disease of the lung. They are: (1) Lobar emphysema; (2) Cystic adenomatoid malformation; (3) Pulmonary sequestration; and (4) Bronchogenic cysts.

The term "congenital cystic adenomatoid malformation of the lung" was introduced into the American literature in 1949 by Chin and Ta 'ng[4] who reviewed ten cases plus adding one of their own. This lesion — once thought to be a hamartoma — lacks certain elements such as cartilage, mucous glands, and alveoli seen with a true hamartoma. It has also been ascribed to a developmental anomaly, or a focal form of pulmonary dysplasia. Recently, electron microscopy has shown features that suggest the lesion is developmental in nature, with a failure of maturation resulting from an insult occurring early

in embryonic life, prior to the onset of cartilaginous differentiation within the lungs.[5]

Pathologically, CCAML is defined as a multicystic mass of pulmonary tissue in which there is proliferation of bronchiolar structures at the expense of the alveoli.[3] Grossly the lesion is firm, rubbery, reddish-brown with cysts of varying size. There are five distinct microscopic features necessary to establish the diagnosis of CCAML.[6] First and foremost is the noted absence of bronchiolar cartilage. This lack of cartilage differentiates CCAML from the other forms of cystic lung disease and from hamartoma. The second feature is the absence of bronchiolar tubular glands. The third is the presence of overproduction of terminal bronchiolar structures without alveolar differentiation. Fourth is an increase of connective tissue, elastic tissue, and smooth muscle. Lastly, there is the presence of tall, columnar, mucinous epithelial cells lining the cystic spaces. Small cysts may be lined by cuboidal epithelium.

CCAML is associated with prematurity, stillbirth, fetal anasarca and maternal hydramnios, and is equally likely to affect males and females. Approximately 25 per cent of patients have associated cardiac lesions or pectus excavatum.[3] Fetal hydrops has been attributed to the obstruction of venous return by the expanding pulmonary mass. Maternal hydramnios is believed to be similar in origin to that occurring with tracheal bronchial obstruction or pulmonary hypoplasia. In these instances, there is a significant loss of lung surface area, or resorptive surface, with a subsequent decrease in the amount of amniotic fluid that is recycled.[5] Also, pressure on the esophagus and excessive secretions by the abnormal lungs may contribute.[7] Hydramnios was observed in our second case.

Seventy-five per cent of afflicted babies exhibit symptoms within the first few hours to days of life manifested as a respiratory distress syndrome resulting from combinations of prematurity, compression, decreased lung tissue, and associated hypoplasia of the remaining lung tissue. Examination often shows a premature hydropic infant with respiratory grunting, sternal and intercostal retraction, and cyanosis. These findings were present in both of our patients. The thorax is usually assymetrical with decreased breath sounds on the same side as the mass, with the mediastinal structures shifted to the side opposite the mass.

Chest x-ray is now routine in the premature infant with respiratory distress and is most helpful in CCAML. As in the two roentgenograms pictured, it most often shows a cystic mass with cysts of varying sizes with a mediastinal shift opposite the mass. Frequently direct communication with the tracheobronchial tree may be apparent on x-ray. The differential diagnosis includes agenesis of the lung, diaphragmatic hernia, staphylococcus aureus pneumonia, and congenital lobar emphysema.

Once the diagnosis has been made, the treatment is thoracotomy and resection, ranging from lobectomy to pneumonectomy. The timing of operative intervention is based on the clinical situation. Both neonates reported here were quite ill and required surgical intervention. Postoperative complications include persistent air leak, infection, and pleural effusion.[8]

In 25 per cent of patients with CCAML respiratory distress is not apparent, and early detection may not be made. If survival occurs without early operation, infection of the malformed lobe is likely to result in a diagnosis of congenital cystic bronchiectasis.[9]

Summary

Congenital cystic adenomatoid malformation of the lung in the newborn is a congenital anomaly of unknown cause. Recent evidence indicates that it may be a developmental anomaly occurring early in the differentiation of pulmonary tissue. Most often it presents as a respiratory distress syndrome in the newborn. The process has certain characteristic radiographic and physical findings which differentiate it from other forms of cystic lung disease with final diagnosis based on the microscopic features. The treatment is thoracotomy and resection. Aggressive diagnosis and therapy are necessary to increase infant survival.

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Tetralogy of Fallot

Cases With Absent Pulmonary Valve

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Tetralogy of Fallot with absent pulmonary valve is a rare congenital cardiac defect. It presents a characteristic clinical picture which includes severe respiratory distress secondary to tracheobronchial compression from aneurysmal pulmonary arteries. The clinical, roentgenographic, electrocardiographic, and angiographic findings of three patients with this lesion are discussed. Success with a new palliative surgical procedure is reported in one of these infants.

TETRALOGY of Fallot is a common cyanotic congenital heart disease. The association of absence of the pulmonary valve with tetralogy of Fallot, however, is a relatively rare congenital cardiac malformation comprising approximately 3-6 percent of autopsy cases of patients with tetralogy of Fallot.[1, 2] There have been 156 cases of patients with tetralogy of Fallot with absent pulmonary valve reported in the literature[3-7] since its initial description in 1847 by Chevers.[8] The pathophysiology of this entity consists of severe pulmonary regurgitation and aneurysmally dilated pulmonary arteries. The clinical problems associated with this anomaly include respiratory distress due to tracheobronchial compression caused by the aneurysmally dilated pulmonary arteries and congestive heart failure secondary to the severe pulmonary insufficiency leading to right ventricular overload. The overall mortality rate for this lesion has been previously reported to be approximately 76-94 per cent with the majority of infants dying during the first year of life.[9, 10] This paper will (1) report the clinical, angiocardiographic, and hemodynamic findings in three patients with tetralogy of Fallot and absent pulmonary valve; (2) discuss the pathophysiology of this lesion with special reference to patients less than one year of age; and (3) report a new surgical palliative procedure for treatment of infants with this lesion.

Case Reports

Case One: A 3232 gm male was born to a gravida 1, para 0, 28-year-old white female with an uncomplicated pregnancy and delivery. Soon after birth the infant was noted to be slightly cyanotic and a loud heart murmur was heard. He was admitted to Wesley Medical Center Newborn Intensive Care Unit at 12 hours of age. Physical examination on admission showed that the infant was cyanotic and in moderate respiratory distress. The precordium was overactive with a right ventricular tap. S_1 was normal and S_2 was covered by a grade 3/6 harsh systolic and a grade 2-3/6 decrescendo diastolic murmur. The murmur was most audible around the upper and mid left sternal border, but was widely transmitted over the entire precordium and back. The liver edge was palpable 3 cm below the right costal margin.

Laboratory findings included arterial blood gases with pH, 7.42; pCO_2, 34 mm Hg; $p0_2$, 25 mm Hg; and oxygen saturation, 47%. The electrocardiogram suggested right atrial enlargement and right ventricular hypertrophy. Chest roentgenograms revealed an enlarged cardiac shadow with aneurysmal main pulmonary artery branches. The vascular markings were decreased peripherally *(Figure 1)*.

Cardiac catheterization with selective right and left ventricular cineangiograms was performed at 1 day of age. The hemodynamic findings are summarized in *Table I*. The right ventricular cineangiogram showed a large and heavily trabeculated right ventricle. There was early opacification of the aorta through a large ventricular septal defect. The pulmonary valve ring appeared stenotic and the pulmonary valve leaflets appeared to be absent. The main pulmonary artery branches were aneurysmal in size *(Figure 2)*. On recirculation the pulmonary veins

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TABLE I
CARDIAC CATHETERIZATION HEMODYNAMIC DATA

			Pressure mm Hg				*O_2 Saturation (%)*					
Case	*Age (Days)*	RA Mean	RV	PA	ΔP	AO	IVC	RA	RV	PA	AO	Qp/Qs
1	1	2	64/0-5	48/8	16	64/48	77	76	83	81	90	1.25/1
2	3	1	68/0-4	—	—	68/32	80	80	81	—	88	—
3	1	4	86/12	72/0	14	—	75	78	76	62	—	1.84/1

RA = right atrium, PA = pulmonary artery, RV = right ventricle, AO = aorta, IVC = inferior vena cava, Qp/Qs = ratio of pulmonary blood flow to systemic blood flow.

drained normally into the left atrium. On selective left ventricular cineangiogram the aorta appeared to arise mainly from the left ventricle, with little overriding.

The hospital course was uncomplicated. The infant was weaned gradually from oxygen. He did not show any signs of congestive heart failure, and was discharged at 5 days of age in good condition.

Follow-up examination revealed normal growth and development. He did require digitalization at 2 months of age, but the primary problem has been repeated respiratory infections in the form of bronchitis and upper respiratory tract infections. He is now 18 months of age and his developmental milestones are normal.

Case Two: A 3500 gm male infant was born to a gravida 1, para 0, 23-year-old white female with an uncomplicated pregnancy and delivery. At birth the infant was noted to be cyanotic and in moderate respiratory distress. He was transferred to Wesley Medical Center Newborn Intensive Care Unit at 6 hours of age. On admission the infant was cyanotic, tachypneic, and dyspneic. The heart was overactive. S_1 was normal and S_2 was covered by a grade 4/6 continuous murmur which was most audible along

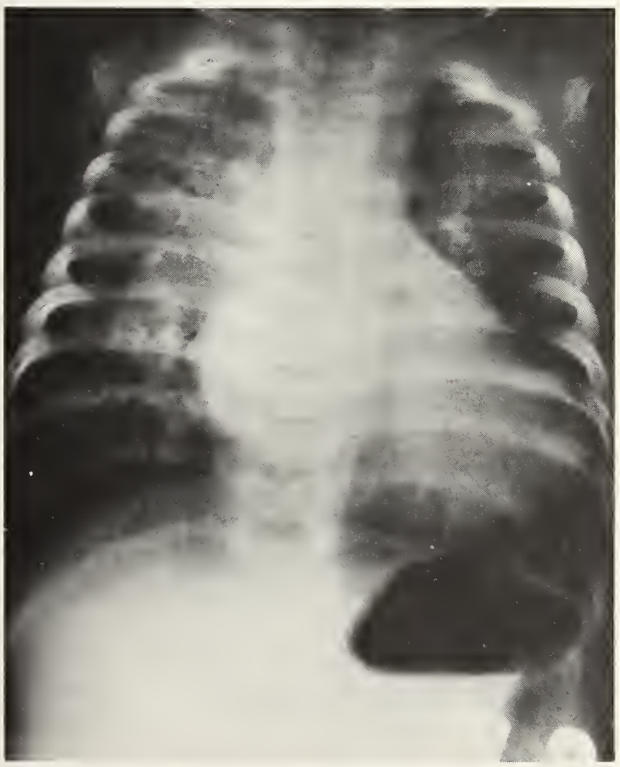


Figure 1. Case One: The chest roentgenogram demonstrates the aneurysmally dilated right pulmonary artery. The heart is enlarged and the cardiac apex is elevated.



Figure 2. Case One: Selective right ventricular cineangiogram, lateral view. Notice that the pulmonary valve ring is displaced superiorly and is indicated by the arrow. The right pulmonary artery seen on end and the left pulmonary artery branches are aneurysmal.



Figure 3. Case Two: Chest roentgenogram taken at 2 days of age. Note the cardiomegaly and prominent right cardiac border created by the dilated right pulmonary artery superimposed on the right atrial shadow. A dilated left pulmonary artery is displaced superiorly and to the left.

the upper left sternal border and transmitted over the entire precordium and to the back. The liver edge was palpable 3-4 cm below the right costal margin.

Laboratory findings included arterial blood gases with pH, 7.36; pCO_2, 39 mm Hg; pO_2, 32 mm Hg; and oxygen saturation, 59%. The electrocardiogram showed right atrial and right ventricular hypertrophy. Chest roentgenograms revealed mild cardiomegaly. The main pulmonary artery branches were markedly dilated and formed shadows on both the right and left cardiac borders *(Figure 3)*. On the echocardiogram, the pulmonary valve echo was recorded, but was not well delineated.

Right and left transvenous heart catheterization was performed at 3 days of age. The hemodynamic findings are summarized in *Table I*. The selective right ventricular cineangiogram revealed an enlarged, heavily trabeculated right ventricle. There was a filling defect at the site of the pulmonary valve leaflets. The main pulmonary artery and the proximal branches were aneurysmal. Early opacification of the aorta was noted due to right-to-left shunting across a large high ventricular septal defect. A selective left ventricular cineangiogram showed that the left ventricle was normal in size.

The hospital course was uneventful. The infant was digitalized and the oxygen was tapered off.

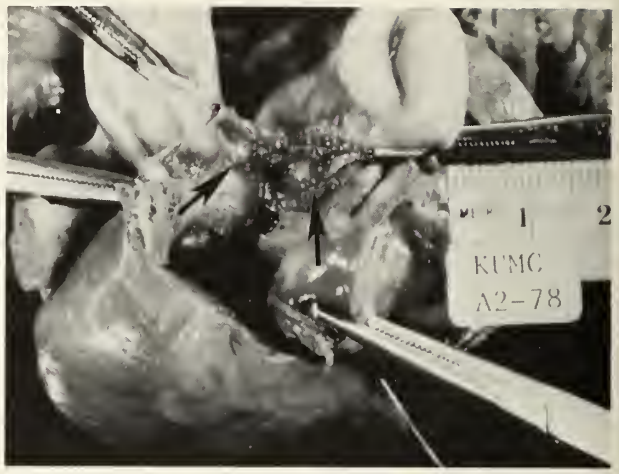


Figure 4. Case Two: Heart specimen showing the right ventricular outflow tract and main pulmonary artery open. Note the rudimentary protrusion of the pulmonary valve indicated by the arrow. The pulmonary valve ring was severely stenotic.

Arterial blood gases revealed persistent hypoxemia, but normal pCO_2 values. He was dismissed at 10 days of age in good condition.

Follow-up revealed that he suffered from repeated episodes of respiratory infections with increasing cyanotic spells and heart failure. He died at 11 months of age after a severe bout of a respiratory tract infection. At autopsy the heart was noted to be enlarged to twice normal size with marked biventricular enlargement. The aorta overrode the large subcristal ventricular septal defect. There was marked right infundibular stenosis. The pulmonary valve was aplastic, consisting of 1-2 mm of a stenotic circumferential rim of gelatinous material *(Figure 4)*. The main pulmonary artery and its branches were markedly dilated with a circumference of 50 mm. The coronary artery pattern was abnormal in that both right and left coronary arteries arose from a single ostium. The lungs were consolidated in all but the left upper lobe. Microscopic examination showed both acute and chronic bronchitis. Post mortem cultures of the lungs grew Klebsiella pneumoniae species. The terminal event was felt to be combined respiratory and cardiac failure.

Case Three: A 2900 gm term Vietnamese female infant was born to a gravida 3, para 2, 25-year-old mother with an uncomplicated pregnancy and normal vaginal delivery. At birth the infant was noted to be cyanotic with mild tachypnea. She was transferred to Wesley Medical Center Newborn Intensive Care Unit for further evaluation.

On physical examination, the infant was found to be mildly cyanotic with mild respiratory distress.

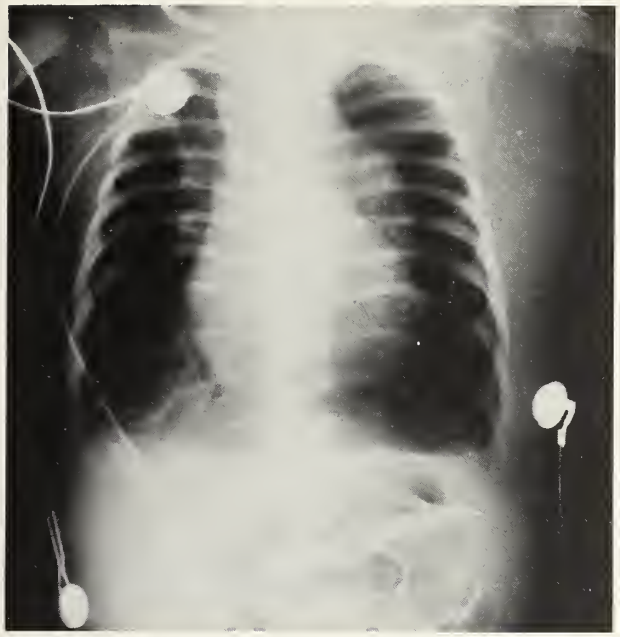


Figure 5. Case Three: Preoperative chest roentgenogram showing hyperinflated lungs. Note the dilated left pulmonary artery superimposed on the upper left cardiac border. Also the cardiac apex is elevated indicating right ventricular hypertrophy.



Figure 6. Case Three: Selective pulmonary artery cineangiogram, A-P view, showing aneurysmally dilated main pulmonary artery and its branches. Note that the main branches taper suddenly toward the periphery.

The heart was overactive and a systolic thrill was palpable along the upper left sternal border. S_1 was normal and S_2 was single. A grade 4/6 systolic and a grade 2-3/6 diastolic decrescendo murmur were most audible along the upper left sternal border and transmitted over the entire precordium, both axillae and the back.

The electrocardiogram showed right ventricular hypertrophy. The chest roentgenogram showed mild cardiomegaly, decreased peripheral pulmonary vascular markings, and prominent pulmonary artery shadows on both sides of the cardiac borders *(Figure 5)*. The echocardiogram was not diagnostic. It showed an enlarged right ventricle with normal mitral and tricuspid valves. A pulmonary valve echo could not be recorded.

Right and transvenous left heart catheterization was done at 1 day of age. The hemodynamic findings are listed in *Table 1*. Selective right ventricular cineangiograms showed an enlarged right ventricle that was heavily trabeculated. There was a right-to-left shunt across a moderately large ventricular septal defect. The pulmonary valve ring was widely patent. The main pulmonary artery and its proximal branches were markedly dilated. Selective pulmonary artery cineangiogram showed massive pulmonary insufficiency *(Figure 6)*.

The infant was dismissed at 5 days of age in good condition without signs of heart failure or respiratory distress.

At 1 month of age the infant was readmitted because of severe respiratory distress due to pneumonia and congestive heart failure. Physical examination revealed a cyanotic infant in acute respiratory distress. She was treated with digoxin and diuretics. Although the congestive heart failure was alleviated, she continued to experience respiratory distress secondary to tracheobronchial compression from the aneurysmal pulmonary arteries. The pCO_2 varied from 95-125 mm Hg and she was begun on a Seimens ventilator. Chest roentgenograms revealed atelectasis of the left lung and right middle lobe. In view of the lack of response to medical therapy, surgery was performed at 6 weeks of age consisting of main pulmonary artery ligation with construction of an ascending aorta to main pulmonary artery shunt with a 4 mm diameter polytetrafluouroethylene (Goretex) shunt. This procedure seemed to help the infant. She was weaned from the respirator two weeks post operatively. She was discharged from the hospital one month following surgery.

Since dismissal, the infant has done well with no further signs of respiratory distress. Chest roentgenograms at 4½ months of age revealed clear lung fields and decreased heart size, but the main pulmonary artery shadow was still somewhat prominent. Currently the infant is 6 months old, growing normally and experiencing no respiratory difficulties and only mild cyanosis.

Discussion

The clinical picture in patients with tetralogy of Fallot with absent pulmonary valve is characteristic and has been well delineated in previous studies[3, 10, 11] and by the three patients in this report. The diagnosis is suspected soon after birth on the basis of a characteristic "to-and-fro" murmur usually most audible at the upper left sternal border and widely transmitted over the entire precordium and back. This "to-and-fro" murmur usually consists of a grade 3-4/6 holosystolic component and a grade 2-3/6 decrescendo diastolic component.[10] The first heart sound is usually normal or increased.[3] The second heart sound is made up of a single aortic component and is usually distinctly audible between the systolic and diastolic murmurs.[3] Phonocardiographic analysis of this characteristic murmur has been recently reviewed.[7] The chest roentgenogram is also characteristic. The heart is commonly moderately enlarged with extremely enlarged main, right and left pulmonary artery shadows, as reported in our three patients. The lateral chest roentgenogram is helpful by showing the large aneurysmal pulmonary artery shadows and usually demonstrates tracheobronchial displacement and compression. Distal to the aneurysmal pulmonary arteries, vascular markings are normal or decreased.[11] However, vascular markings can be increased in patients with predominant left-to-right shunts.[3] The electrocardiogram usually shows marked right ventricular hypertrophy. In our experience, the echocardiogram is not diagnostic. Recently, however, it has been suggested that abnormal echoes in the right ventricular outflow tract representing dense subpulmonary conal muscle and rudimentary pulmonary valve tissue can be demonstrated.[4]

Cardiac catheterization is the definitive procedure for confirming the diagnosis of tetralogy of Fallot with absent pulmonary valve. Right ventricular pressure in this lesion is always elevated and equal to systemic pressure. The gradient across the pulmonary valve can be quite variable, depending on the amount of stenosis present at the pulmonary annulus. Infundibular stenosis and supravalvular stenosis can also be present.[11] The diastolic pressure in the pulmonary artery is usually the same as the right ventricular diastolic pressure due to the free pulmonary regurgitation. The angiocardiogram commonly demonstrates a dilated right ventricle and biventricular shunting across a large ventricular septal defect. Prolonged emptying time of contrast material from a right ventricular injection is due to massive pulmonary regurgitation. In our patients, the lateral view of the right ventricular cineangiogram was very helpful in visualizing the stenotic pulmonary annulus with the absence of pulmonary valve leaflets *(Figure 2)*. Selective pulmonary arteriography demonstrates the location and extent of the pulmonary artery aneurysmal dilation.

The prognosis for infants with tetralogy of Fallot with absent pulmonary valve depends on the onset of symptoms. The mortality rate is high in those infants who develop marked respiratory distress and congestive heart failure in early infancy. Those with minimal respiratory symptoms follow a course similar to infants with common tetralogy of Fallot.[11] The severe respiratory complications are clinically manifested by hypercarbia, tachypnea, development of obstructive emphysema, and atelectasis.[3] Infants who survive the neonatal period frequently have repeated bouts of bronchopneumonia as exemplified by Cases One and Two in our report. Often the symptoms caused by tracheobronchial compression by the aneurysmal pulmonary arteries resolve spontaneously at the age of one year.[11] This is due to maturational changes in the bronchi with increased caliber and cartilaginous support of the bronchi.[12]

Treatment of infants with tetralogy of Fallot and absent pulmonary valve depends on the severity of the symptoms. In those patients with mild respiratory symptoms and minimal congestive heart failure, aggressive medical therapy is indicated. Respiratory tract infections should be treated vigorously or perhaps prophylactically with antibiotics to prevent severe cardiorespiratory decompensation or death from such an infection, as in Case Two. Constant positive airway pressure may be helpful during an acute period of decompensation.

In those infants with severe respiratory distress and intractable heart failure, surgical intervention is necessary in addition to aggressive medical therapy. Many different surgical palliative procedures have been proposed including aneurysmorraphy,[11] aneurysmectomy[11], and transection of the aneurysmal right pulmonary artery with anastamosis to the superior vena cava[13] or distal right pulmonary artery via a Dacron conduit.[14] Experience with each of these procedures has been small and the overall results somewhat disappointing.

The pathophysiology of this lesion is based on the increased stroke volume of the right ventricle, which is secondary to the gross pulmonary insufficiency. Thus, with each systole a greater than usual volume of blood is ejected into the pulmonary arteries. During diastole some of this volume flows back into the

right ventricle and the cycle is repeated. This process leads to aneurysmal dilatation of the proximal pulmonary arteries and congestive heart failure. These arteries then compress the proximal tracheobronchial tree with resultant obstructive emphysema and atelectasis.

The surgical procedure in Case Three was designed to eliminate the pulmonary insufficiency by ligating the main pulmonary artery. An aorta-to-pulmonary-artery shunt was then constructed beyond the point of ligation. The shunt provides enough pulmonary blood flow to maintain acceptable arterial oxygen saturation. The advantages of this procedure are that it is easier to perform in the critically ill neonate and the normal pulmonary artery anatomy is not radically altered as in previously described operations. Hopefully, this will simplify later total correction.

Definitive correction of this lesion has been accomplished in a number of older children not severely affected in early infancy with respiratory distress and heart failure.[15-17] Total definitive correction in severely affected neonates has been suggested utilizing profound hypothermia, but to date there has been no report of success using this approach.[18]

The clinical, roentgenographic, electrocardiographic, and angiographic findings of three patients with tetralogy of Fallot with absent pulmonary valve have been discussed. The main problem seen in this entity is severe respiratory distress and congestive heart failure manifest in early infancy. The diagnosis in the newborn is suspected when a loud "to-and-fro" murmur is audible over the chest with a chest roentgenogram showing anerysmal pulmonary artery shadows. A new surgical palliative procedure consisting of ligation of the pulmonary artery and ascending aorta to main pulmonary artery shunt was utilized successfully in one patient.

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Malaria

An Unusual Cause of FUO in Kansas

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THE DIFFERENTIAL diagnosis of fever of unknown origin (FUO) is long and complex. With the recent immigration of thousands of Southeast Asian refugees to the United States, our differential needs to be broadened to include several of the tropical diseases common to that area. We present here a case of FUO in a Vietnamese boy.

Mobility of population has resulted in a concurrent mobility of disease. Physicians in the United States can no longer overlook the tropical diseases in the differential diagnosis of fever of unknown origin. The case of a Vietnamese child with malaria, treated at a Wichita hospital, is presented and therapeutic regimens discussed.

Case Report

A 7-year-old Vietnamese male presented to the emergency room of St. Francis Hospital, Wichita, on March 7, 1980, with a two-hour history of fever, shaking chills, and repeated vomiting. He had been seen at the local health department that afternoon where a tuberculin skin test was applied; he was referred to the emergency room. The boy had arrived in this country two days earlier with his mother and four healthy siblings. The father, who had been in the United States for four months and could speak some English, stated that he had a similar illness following his arrival in this country, but could not remember what he had been told.

The patient had been in good health until that afternoon. Little past medical history was available except that he was on no medication and had no known allergies.

Physical examination revealed a thin, lethargic, and toxic appearing 7-year-old. His temperature was 39.3C at 3 PM; pulse, 140; and respirations, 24. Blood pressure was 100/50. There were no meningeal signs. Positive findings included: a moderately injected posterior pharynx with small vesicles, numerous dental caries, and a soft grade 1/6 systolic murmur heard at the left sternal border without radiation. S_1 and S_2 were normal. The abdomen was slightly tender to palpation, but the liver and spleen were not enlarged. Other results of examination were normal.

The original blood smear was thought to be negative for any parasites. Hemoglobin was 11.3; hematocrit, 33.3; WBC, 7,400 with 23 bands, 40 segmented, 22 lymphocytes, 11 monocytes, 1 eosinophil, 1 basophil, and 2 metamyelocytes; and platelets, 129,000. Urinalysis showed 1+ ketones and 10 RBCs/hpf; and sedimentation rate was 103. Electrolytes and all other blood chemistries were normal. Chest x-ray revealed a prominent spleen, borderline cardiomegaly, and a slight increase in upper lobe vascularity.

By 4 PM temperature was 39.4C. Bacterial cultures of blood and throat were obtained, as well as sputum for acid fast bacilli testing and a throat culture for viruses. These subsequently all proved negative. EKG was normal. Thick and thin smears for malarial parasites were obtained. By 7 PM his temperature had dropped to 36.5C and the patient appeared completely well.

On the following day, the pathologist reported the presence of malarial parasites — thought to be P. vivax — in the smear. Hemoglobin had dropped to 10.3 and then to 9.7 one day later. G6PD screen showed normal activity. B_{12} was 685 and folate 14.3, both normal. Immunoglobulins revealed an active inflammatory pattern with an IgG of 1777; IgA, 239; IgM, 242. Serum for antibody testing was sent to the state laboratory.

Chloroquine was begun on the day following admission at an initial dose of 10 mg/kg orally; this was followed at six hours by 5 mg/kg, another 5 mg/kg at 24 hours, and a final 5 mg/kg at 48 hours. Primaquine was begun on March 10 at a dose of 0.25-0.3 mg/kg/day divided into two doses for 14 days. Stool for ova and parasites could not be obtained. The tuberculin test showed 15 mm of induration on March 10. It was unclear if the patient had previously had a BCG vaccination. He con-

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tinued afebrile after the evening of March 7 and was discharged on March 11, apparently well. No parasites were seen in the peripheral smear after March 8. The state laboratory reported the antibody titers to be P. vivax, 1:1024; P. falciparum, 1:64; and P. malariae, 1:64.

Discussion

Malaria must be considered in the differential diagnosis of FUO in the Southeast Asian population now arriving in the United States as well as in unprotected travelers. Several excellent reviews are available in textbooks and journals on the clinical course and life cycles of the various forms of malaria.[1-4] Following is the latest information available for treatment and prophylaxis from the Center for Disease Control.

The Center for Disease Control reported the incidence of malaria in travelers to various parts of the world from 1970-1976. Most cases were contracted in Africa (48.7%), followed by Central America and Mexico (20.6%), and Asia (19.9%). The largest number of cases were due to P. vivax (54.8%) and P. falciparum (31.6%). Of the cases acquired in Africa, 49.5 per cent were P. falciparum and 30.6 per cent were P. vivax. In Asia, Central and South America, P. vivax (78%) was the most common with P. falciparum (14%) a distant second.[5]

The problem of chloroquine-resistant forms of P. falciparum is becoming important. Resistance to this drug has been identified in Southeast Asia, Indonesia, Philippines, Papua, New Guinea, India, Bangladesh, Burma, Panama, and South America.[5, 6] Until recently, Africa was spared from resistant forms of P. falciparum, but several cases have been reported in East Africa and this may become significant in the future.[7]

The therapeutic regimen for malaria due to P.

TABLE I
DRUG THERAPY OF MALARIA BY SPECIES*

Species	*Drug*	*Adult*	*Children*
1. P. vivax P. ovale	Chloroquine	600 mg base initially p.o., followed by 300 mg base at 6°, 24°, 48°	10 mg/kg initially p.o. (max 600 mg) followed by 5 mg/kg at 6°, 24°, 48°
	plus		
	Primaquine	15 mg/day for 14 days p.o.	0.3 mg/kg/day (max. 15 mg) for 14 days p.o.
2. P. malariae	Chloroquine (alone)	(see above)	(see above)
3. P. falciparum	1) quinine	650 mg tid for 3 days	25 mg/kg/day ÷ 3 doses for 3 days
Uncomplicated attack:	*plus*		
	Pyrimethamine	25 mg bid for 3 days p.o.	<10 kg, 6.25 mg/day p.o. 10-20 kg, 12.5 mg/day 20-40, 25 mg/day for 3 days
	plus		
	sulfadiazine	500 mg qid for 5 days p.o.	100-200 mg/kg/day ÷ 4 doses for 5 days p.o.
	2) quinine	(see above)	(see above)
	plus		
	tetracycline	500 mg qid for 7 days p.o.	20 mg/kg qid for 7 days p.o.
For severe illness	quinine dihydrochloride	600 mg in 300 ml NS IV over 1 hr, repeat in 6-8 hours (Max 1.8 gm/day)	25 mg/kg/day ½ dose in 1 hr infusion followed by ½ dose in 6-8 hrs.

* From The Center for Disease Control.

TABLE II
CHEMOPROPHYLAXIS FOR MALARIA BY AREA*

Area	*Drug*	*Adult*	*Children*
1. Asia	(Fansidar®)		6-11 mo. — 1/8 tab
Central and South America	Pyrimethamine (25 mg)	1 tab p.o.	1-3 yr. — 1/4 tab 4-8 yr. — 1/2 tab
	plus	weekly	9-14 yr. — 3/4 tab
Caribbean	Sulfadiazine (500 mg)		p.o. weekly
2. Africa	Chloroquine or Fansidar®	300 mg base p.o. once weekly (see above)	5 mg/kg p.o. once weekly

* From The Center for Disease Control.

vivax, P. ovale, and P. malariae utilizes chloroquine phosphate (Aralen). This will eradicate the erythrocytic phase of the disease. Side effects of chloroquine are few, but may include headaches, transient visual disturbances, nausea, and pruritus. Since relapses of malaria due to P. vivax and P. ovale may occur due to the continued presence of the exoerythrocytic phase, primaquine should be added on the second day of therapy with chloroquine. One should screen for G6PD before starting primaquine as severe red-cell hemolysis may occur in those with a deficiency. If G6PD deficiency is present, primaquine can be given during a period of eight weeks at 45 mg weekly for adults and 0.75 mg/kg weekly for children.

It must now be assumed that any P. falciparum is resistant to chloroquine, and because of the increased mortality in this form (5.7%),[5] therapy should be initiated as soon as identification of the parasite is made. For uncomplicated attacks two regimens are available, each yielding approximately a 96-per cent rate of cure *(Table I):* (1) Quinine sulfate *plus* pyrimethamine, *plus* sulfadiazine; or (2) Quinine sulfate *plus* tetracycline. For those seriously ill with the complications of severe vomiting, acute renal failure, cerebral malaria, or pulmonary edema, quinine dihydrochloride may be given intravenously along with careful fluid and electrolyte therapy.

Chemoprophylaxis for travelers to endemic areas is best accomplished with Fansidar, a combination of pyrimethamine (25 mg) and sulfadiazine (500 mg), which is not available in this country but can be obtained over-the-counter in most endemic areas *(Table II)*. Ideally the patient should begin taking the drug one week before arrival in the area and continue through the period of exposure and for four to eight weeks after leaving. Pyrimethamine is contraindicated during pregnancy.

In the past, adequate prophylaxis for travel to Africa could be achieved with chloroquine. Since this drug will not prevent the exoerythrocytic phase and relapses, primaquine can be added the last two weeks of, or following, the course of chloroquine. The addition of primaquine should be considered in those who may have had heavy exposure. If the resistance to chloroquine among African P. falciparum increases, Fansidar will be come the prophylactic agent of choice.

No longer can the United States physician ignore the tropical diseases of the world. Malaria must be considered in the differential diagnosis of fever of unknown origin in the population arriving in the United States from Southeast Asia and more recently from Haiti. A high index of suspicion and prompt medical care may prevent some of the complications and mortality associated with this disease. It is strongly recommended that travelers to endemic areas avail themselves of appropriate chemoprophylaxis to prevent the considerable morbidity associated with malaria.

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Regional Perinatal Care

Kansas/1979

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THE GOAL of a regional perinatal care program is to provide high level care to every citizen regardless of geographical location. For Kansas, this began in 1973 with the formation of a statewide Perinatal Steering Committee composed of obstetricians, pediatricians and family practice physicians, along with public health personnel, nurses, and interested health care providers. The initial objectives were:

1. To establish and maintain an effective transport system to link patients to appropriate level care centers;
2. To designate Level I and Level II care centers, assist in their development and provide support from the two Level III centers (UKSM-KC and Wesley Medical Center, UKSM-Wichita);
3. To develop programs for education, updating, and training in high risk obstetrics and neonatology and allied health care services;
4. To provide 24-hour consultation and patient evaluation services;
5. To develop audio-visual aids to facilitate education and training of health care providers;
6. To create educational programs for laity of childbearing age;
7. To coordinate and develop resources for after-care and follow-up of infants and mothers;
8. To establish counseling centers for antenatal diagnosis; and
9. To create a data system for obtaining uniform perinatal information statewide, and to establish a perinatal clinical research laboratory facility.

In March of 1974, Howard Fox, M.D., of UKSM-KC, and Daniel K. Roberts, M.D., Ph.D., Wesley Medical Center, UKSM-Wichita, were appointed co-directors. Funding was obtained from the Kansas Regional Medical Program. The Program continues to expand to meet the goals. Within the past year the antenatal diagnosis facility at Wichita was opened not only for counseling, but also for processing of fetal tissue cultures. Significant steps have been taken to implement data collation with computer application.

The regional Perinatal Care Program in Kansas has achieved a significant degree of effectiveness despite problems relating to a diversity of geographical situations and a low population density. The present status of the Program is reviewed and future objectives defined.

The Program functions in four major areas:

1. Patient services, including transportation, hospitalization, and professional care;
2. Education, organization, and consultative services;
3. Post discharge evaluations and follow-up care; and
4. Administration for statewide coordination.

From the beginning, the dedicated health care providers and the citizens of Kansas have supported the concept of regional perinatal care.

Patient Services

For regionalization in Kansas to have relevance to its approximately 2,250,000 citizens, the health care services need to be available to all, even though the area includes 82,264 square miles. The overall population density for Kansas is about 27.5 persons per square mile. This density is considerably below that of the Northeastern states — for example, New Jersey has 953.1 persons per square mile. However, Alaska (with 0.5 persons per square mile) and 12 other states have population densities lower than Kansas. Most of the population in Kansas is concentrated east of Wichita and Hutchinson. Consequently, the transport needs for Western Kansas are different from those for Eastern Kansas.

Neonatal transports outnumber maternal transports by about 3:1. Future goals include increasing the number of maternal transports relative to the

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TABLE I
PERINATAL TRANSPORTS — UKSM-WICHITA

Year	*Neonatal*	*Maternal*	*Total*
1976	257	68	325
1977	294	115	409
1978	304	99	403
1979	337	95	432
	1192	377	1569

Perinatal Transport by Vehicle

Year	*MPICU**	*Air*	*Private*
Last ½			
1977	93	98	71
1978	135	181	99
1979	141	227	64
	369	506	234

* Mobile Perinatal Intensive Care Unit.

neonatal, or at least increasing the percentage of complicated obstetrical cases delivered at the Level II and III centers. There may be a slight, but not significant, increase in the percentage of deliveries done at the larger hospitals with 500-1000 deliveries per year since 1974, but this change does not necessarily reflect an increase in high risk pregnancies *(Table I)*. A calculation of the percentage of deliveries in facilities with less than 500 and 1000 deliveries per year compares favorably with the 1970 American College of Obstetrics and Gynecology National Study of Maternity Care *(Table II)*.

Maternal risk factors are recognized by many Kansas physicians and referred for similar diagnoses with frequencies comparable to several other smaller series[1-4] *(Table III)*.

TABLE II
PERCENT OF DELIVERIES IN KANSAS FROM HOSPITALS WITH LESS THAN 500 AND 1000 DELIVERIES/YEAR

Year	*< 500/Year*	*< 1,000/Year*	*Total Births*
1970 ACOG survey*	50.0%	75.0%	
1972 Kansas†	46.9%	61.3%	33,176
1974 Kansas†	43.4%	58.4%	32,712
1976 Kansas†	39.3%	58.1%	35,278
1978 Kansas†	39.2%	56.2%	36,581

* National Study of Maternity Care, Chicago, ACOG, 1970.

† Kansas State Department of Health and Evironment, Bureau of Registration and Health Statistics 1978.

TABLE III
MATERNAL TRANSPORTS (to 5/1/80)

Premature labor	134
Premature rupture of membranes	79
Premature labor/PROM	52
Pre-eclampsia	43
Bleeding	25
Premature labor/bleeding	17
Placenta previa	11
Premature labor/infection	10
PROM over 48 hours	10
Placenta abruption	8
Eclampsia	8
Prolonged labor	8
Premature labor/diabetes	6
Fetal distress	5
Pre-eclampsia/premature labor/diabetes	4
Fetal death inutero	4
Pre-eclampsia with partial abruption	3
Postpartum complication	3
Postpartum bleeding	2
Bleeding/pre-eclampsia/premature labor	2
Premature labor/ruptured uterus	2
Pregnancy induced hypertension	2
Other	14
Total	453

Statistical data support benefits of regionalization for Kansans. Since the Kansas Regional Perinatal Care Program began in 1974, the neonatal mortality rate for Kansas has decreased from 12.0 to 8.3/1000 live births in 1978 *(Table IV)*. This trend has been demonstrated with other programs, especially that of Usher.[5] UKSM-Wichita, Wesley Medical Center has primary coverage for regions I, II, and III *(Figure 1)*. This large area of coverage relative to Kansas City has been possible because of a multifaceted transport system.

The complex task of facilitating patient care with several hundred miles separating patient and physician from necessary expensive equipment has been a challenging one. The concept of patient transport is

TABLE IV
KANSAS NEONATAL MORTALITY RATE SINCE PERINATAL CARE PROGRAM

1974	12.0
1975	10.1
1976	10.3
1977	10.1
1978	8.3

Kansas State Department of Health and Environment, Bureau of Registration and Health Statistics.

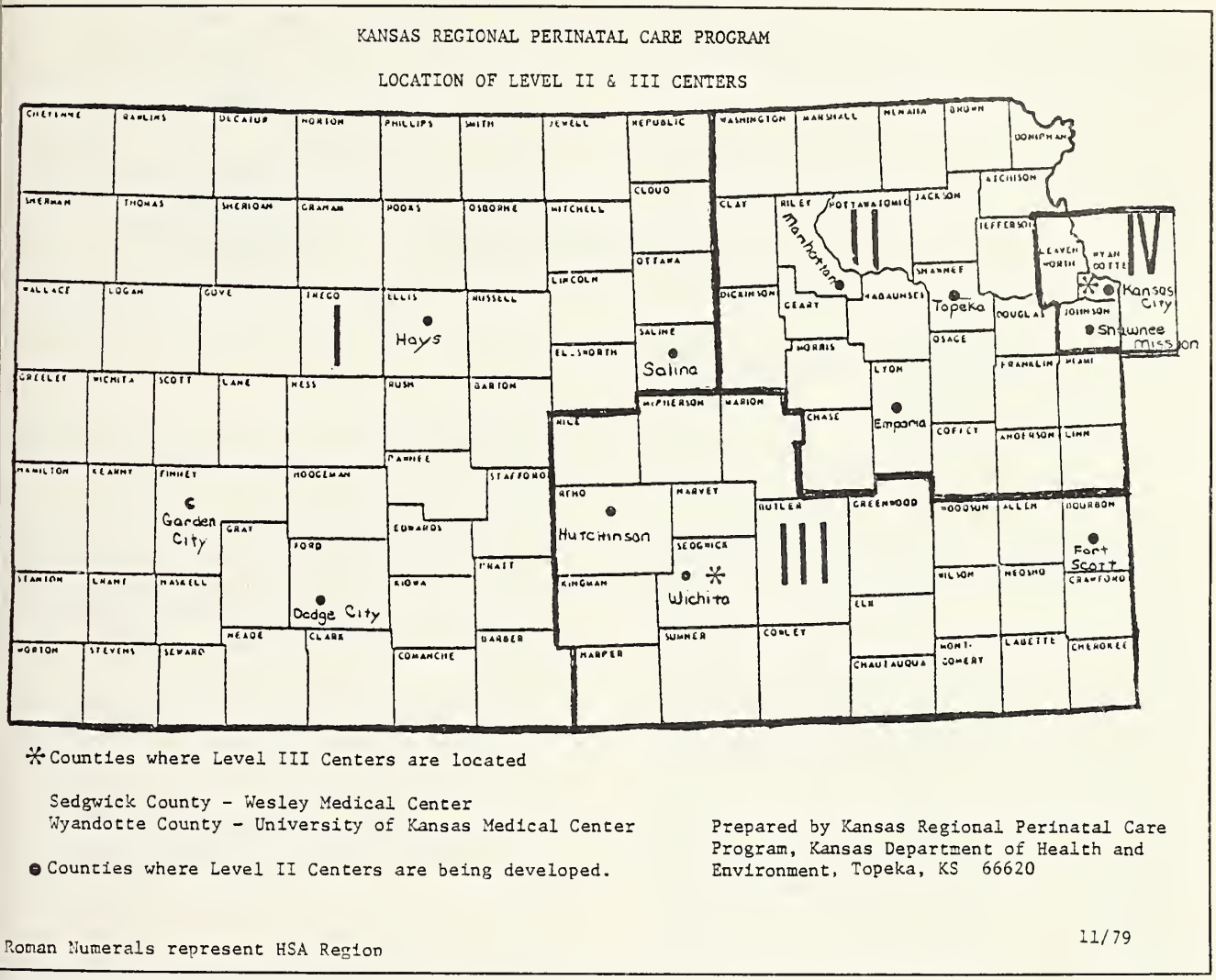


Figure 1

certainly not new and, in fact, occurs daily when husband, parents, or friends put an expectant mother in a car and take her to the hospital. The more critical a patient's condition, the more complex and expensive is the equipment needed to provide necessary information and administration of health care directed by the physician or other health care providers. In 1973, the first transport system for Wesley Medical Center was comprised of R. A. Nelson, M.D., a transport incubator, and a Kansas State Highway Patrol squad car. The following year, a van was obtained and equipped to handle intensive care neonatal and maternal patients, including provisions for emergency cesarean section should it become imperative. Before long, the transport van — "Betty" — was celebrating its 100th mission. However, the need for greater speed was soon apparent, particularly during intervals of inclement weather, and in 1975 the transport fleet was increased by acquisition of a Beechcraft Queen Air backed by a Piper Navaho called into service three or four times per month. These aircraft provided rapid transportation for the critical neonate and parturient. Increased use of the aircraft, a demand to extend the range of transports, and the need for pressurization fostered acquisition of a turbo-prop Beechcraft King Air, and the Queen Air was retired. The transport mechanism was then expanded to include trauma and critical care patients. In the summer of 1979, a Cessna Citation (twin-engine jet) was added to the air fleet and has proved useful for trips to Southwestern Kansas and extended inter-hospital transfers. In the meantime, "Betty" was retired, replaced by "Betty II," a GMC unit converted to transport capability.

In July 1970, Project MAST (Military Assistance to Safety and Traffic) had become available through

the Department of Defense in cooperation with the Department of Health, Education and Welfare, and their services have been used in Kansas through the cooperation of the facility at Ft. Riley. As the needs of the perinatal transport system became more precisely defined, helicopter feasibility was considered and an intensive review of other programs was implemented. Early in 1979 the Perinatal Program at Wichita combined with the Critical Care Transport Program to support an Alloeuette helicopter which was placed on 24-hour duty with a three-minute response time. This unit was selected for its history of excellent service and fuselage width, which could accommodate two stretcher patients (adults). The fixed base operator at Comotara Airpark in Wichita provides pilot service, aircraft maintenance, and 24-hour crew quarters at the airport. The operation would not be possible without their willingness to assume the burdens of maintaining complex equipment and highly skilled flight personnel on a 24-hour basis. Medical crew training is an extensive, ongoing endeavor of which the Perinatal Program is very proud. Their curriculum and certifications deserve special mention and include RN, EMICT, and ongoing critical care experience in all the critical care units of the Wesley Medical Center. There exists an urgent need for standardization of equipment and personnel training in the aeromedical services.

All transports comply with the Patient Transfer Guidelines[5] approved by the American College of Emergency Physicians, including physician-to-physician communication, plan for appropriate pre- and intra-transfer care, preparation of pertinent medical records, prearranged transfer care plans for evaluation of specific medical needs, and specific management and transfer responsibility.

Education

The most elaborate of regional systems fails if the first line health care providers — referring physicians — fail to identify potential patient problems. It is therefore essential that these alert and responsible physicians and nurses have the opportunity to obtain the latest medical information. The education of these professionals has been a major effort for the Program. With physician direction and support, RNs with special training and interests in education, transport, and intensive care of critical patients along with registered social workers and respiratory therapists comprise the nucleus of the medical support outreach team. During 1979, 43 outreach education programs were staffed by UKSM-Wichita, Regional Perinatal Care Program. Twenty-five of these utilized staff physicians including obstetricians, neonatologists, pediatricians, and a geneticist. The medical center has also hosted several physicians for brief, specialized training. Students at several schools of nursing outside of Wichita have received some of their obstetric and newborn experience through the perinatal division. The perinatal division has also sponsored two conferences per month relating to patient care. Inservice training for personnel to assist with labor and delivery as well as newborn services is organized and staffed by the perinatal division. The division staff members participate in many national and local organizations that foster care for the perinates; they also serve in educational and advisory positions.

Outreach education will continue to be a major part of the Program's goals as outlined by the 1980 objectives. These include the following:

1. Maintaining communications with Directors of Nursing and education coordinators of outlying hospitals to insure awareness of programs available;
2. Continued willingness and obligation to visit outlying facilities to foster communication and patient care;
3. Expanded use of education for both didactic and clinical instruction for health care personnel;
4. Increased use of the Prenatal Testing Center along with consultation; and
5. Increased feedback from follow-up clinic evaluations of infants treated through the Perinatal Program.

Discharge and Follow-Up

Last year 362 patients were seen as follow-up from previous Perinatal care transports. Six outreach clinics were held throughout Kansas. Plans to incorporate developmental studies with growth assessments are being applied. This data will then be coded and evaluated in the near future.

Administration

Since 1974, it has become increasingly apparent that a systematic method for organizing the data is essential. Several data systems have been developed or adapted throughout the country, and these systems have been extensively reviewed for their potential application to the Kansas Regional Program.

It has been established that approximately 25 per cent of hospital expenses are related to such factors as record keeping, record processing, and data reporting; therefore, any system utilized by the Kansas Regional Perinatal Program must be designed to minimize time and expense. Acquisition of data requires forms, and there is an obligation for those

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Metoclopramide: A Promising Old Drug

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Metoclopramide (methoxychloroprocainamide) (Reglan) is an analog of procainamide that was synthesized in France in 1963. It suppresses nausea and vomiting and enhances upper intestinal motility with essentially no effects on the heart. It has been available for the past 12-15 years in most countries of the world. The Food and Drug Administration (FDA) has judged the injectable form effective for placement of intestinal tubes in the duodenum and for radiographs of the small bowel. The injectable form of metoclopramide is currently available. FDA approval of the oral tablet form for diabetic gastroparesis can be anticipated.

Clinical Uses

The primary use of metoclopramide is for nausea and vomiting *(Table I)*. A number of studies of this application have been reviewed.[1-3] Many are controversial because they have not been conducted as controlled clinical trials.

Other clinical indications include gastroesophageal reflux and slow gastric emptying. A group of United States investigators performing a controlled clinical trial report metoclopramide is useful for reducing the symptoms of patients with gastroesophageal reflux. Seventeen patients taking placebo experienced a 30 per cent reduction in symptoms, while the 14 patients on metoclopramide experienced a 58 per cent reduction in symptoms ($p<0.05$).[4] About half of the patients experienced greater than 60 per cent reduction in symptoms with metoclopramide while in others it had no therapeutic effect. Metoclopramide may be most effective in those patients with mild or moderate gastroesophageal reflux.

In another controlled clinical trial in the United States, investigators found that metoclopramide was effective in reducing the symptoms of slow (delayed) gastric emptying. The patients had several causes of slow gastric emptying — post-vagotomy stasis, diabetic gastroparesis and idiopathic stasis — but the drug-treated and placebo groups were comparable. The 14 patients treated with placebo had a 32 per cent reduction in symptoms, while the 14 patients on metoclopramide experienced a 60 per cent reduction in symptoms ($p<0.01$).[5] There are additional United States studies (multicenter controlled clinical trials which have not been published to date) that are consistent with these positive results in patients with post-vagotomy stasis and diabetic gastroparesis. These domestic results are similar to previous findings.[2]

Temporary states of slow gastric emptying may

TABLE I
GASTROINTESTINAL INDICATIONS FOR METOCLOPRAMIDE

1. Nausea and vomiting
2. Gastroesophageal reflux
3. Slow gastric emptying
 a. Post-vagotomy stasis
 b. Diabetic gastroparesis
 c. Idiopathic stasis
4. Small intestine
 a. Tube placement
 b. Radiographs

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occur in many patients, especially in the postoperative period and in pregnant women near term. In one study with pregnant women in labor, the control patients emptied 350 ml of an initial 750 ml of intragastric water in two hours, while women who received metoclopramide emptied 650 ml ($p<0.001$).[6] The drug had no other detectable effect on the mother, the baby or the labor process.

Metoclopramide reduces the time it takes to place a small intestinal biopsy tube near the ligament of Treitz. In 25 control patients, it took the physician 23 minutes to place the biopsy tube, while it took only 11 minutes in 25 patients receiving 10 mg of metoclopramide intravenously ($p<0.01$).[7] The fluoroscopy time was also reduced by one-half. This study suggests metoclopramide would be useful in placing decompression or feeding tubes in the duodenum.

Metoclopramide has been used for a number of years by European radiologists for small bowel radiographs. The time to complete the examination is reduced and the radiographs are of good quality.[1, 2] Metoclopramide also reduces nausea and abdominal distress that may be associated with the rapid oral electrolyte overload method of cleansing the colon for barium enemas or colonoscopy ($p<0.01$).[8]

Human Pharmacology

With 10 mg of metoclopramide intravenously, given over one to two minutes, the onset of action is within three to five minutes. The onset of action with 10 mg orally is about 30-60 minutes. About 20-30 per cent of an oral dose is altered by the intestine or liver (first pass effect). The half life of the drug is about four to six hours, and about 75 per cent of the drug is excreted in the urine. About half of the drug in the urine is in the free form. Sulfate and glucuronide conjugates have been detected in the urine. About 5 per cent of the drug is found in the feces and 20 per cent is extensively metabolized to a number of compounds[9, 10] *(Table II)*. Since renal excretion is the major elimination route for metoclopramide, patients with a significant reduction in renal clearance may require smaller doses of the drug. The usual oral dose is 10 mg four times/day, 30 minutes before meals and at bedtime. Doses up to 80 mg/day have been used, but a higher incidence of side effects may be observed.

Mechanism of Action

While the exact mechanism of action of metoclopramide in man is unknown, its effects are best understood in terms of its potent dopamine antagonism. Dopamine has an important role in the central nervous system (CNS) and autonomic nervous system.[1, 2] Increased dopamine levels in the CNS result in nausea and vomiting in animals. Apomorphine, a narcotic antagonist, also causes vomiting by stimulating dopaminergic receptors in the chemoreceptor trigger zone in the brain. Metoclopramide antagonizes the dopamine effect in these areas to provide an antiemetic action.

TABLE II
PHARMACOKINETICS OF ORAL METOCLOPRAMIDE

Onset of action	30-60 min.
Serum half life	4-6 hrs.
Excretion	
Urine	75%
Free	1/2
Conjugated	1/2
Feces	5%
Metabolized	20%

Prolactin secretion is normally suppressed by dopamine. Metoclopramide blocks this effect; prolactin is released and its level rises in the serum. Metoclopramide is one of the most potent releasers of prolactin and may thereby cause breast tenderness and galactorrhea.

In Parkinson's disease, the extrapyramidal tremors are enhanced by insufficient dopamine concentrations in the basal ganglia. Levodopa (L-DOPA), a dopamine precursor, is used as a therapeutic agent to reduce Parkinsonian tremors. Metoclopramide is a dopamine antagonist at this site, and may cause or worsen Parkinsonian tremors.

In experimental animals, dopamine infusions result in relaxation of the stomach. The stomach distends and the rate of gastric emptying is slowed. When this effect is sustained with a constant infusion of dopamine, the relaxing effect is blocked by metoclopramide, with reduced distension and increased rate of emptying.[11]

In addition to the pharmacological studies, a number of physiological studies have been carried out, in man as well as animals. Metoclopramide strengthens esophageal contractions and probably increases the rate of propagation. The resting pressure of the lower esophageal sphincter is increased in a dose-dependent manner, but low resting pressures may not be much increased. Metoclopramide also increases the rate of emptying of liquid and solid meals from the stomach, especially when the initial emptying rate is slow.

The pharmacology of metoclopramide is not completely explained by the dopamine hypothesis be-

cause the metoclopramide effect is blocked by anticholinergics such as atropine. Muscarinic receptors are probably involved and perhaps there are two different dopamine receptors.[12]

Side Effects

Most of the side effects of metoclopramide are associated with the CNS and may be due to blocking the normal effects of dopamine. The most common side effect of metoclopramide, which occurs in 4-10 per cent of patients, is a feeling of drowsiness or lethargy.[2, 3] Patients fall asleep while reading the paper or watching television. A feeling of anxiousness or agitation is less common. In less than 1 per cent of patients, a Parkinsonian tremor may develop. This is usually experienced by the very young and the elderly following high doses over a long period of time. Breast tenderness and swelling with galactorrhea may result in some patients.

The side effects of metoclopramide are generally not serious and subside within a few days when the drug is discontinued. Since most effects of metoclopramide can be blocked by anticholinergics, atropine is effective and has been used. The use of metoclopramide throughout the world for a number of years suggests its relative safety. Doses of 600 to 800 mg have been taken acutely in suicide attempts without serious side effects.

Contraindications

The recognized contraindications are few and can be divided into absolute and relative contraindications *(Table III)*. One absolute contraindication is the presence of a pheochromocytoma. Three patients with this disorder experienced hypertensive crises after receiving metoclopramide. This implies that metoclopramide released epinephrine and norephinephrine from the tumor along with other pressor or mediators. As with any other drug, a sensitivity reaction is another absolute contraindication. Since metoclopramide is chemically similar to procaine and procainamide, the possibility of cross sensitivity should be considered. Patients with complete intestinal obstruction should probably not receive the drug because increased peristalsis might contribute to a perforation. In frank intestinal perforation, metoclopramide might lead to further contamination of the peritoneal cavity.

Relative contraindications include Parkinson's disease which would probably be exacerbated by the dopamine antagonistic actions of metoclopramide. There might be an additive effect with certain sedatives, especially in those patients prone to the drowsiness side effect of metoclopramide. In patients receiving therapeutic doses of anticholinergic drugs, metoclopramide may not be effective, as anticholinergics block the pharmacological effects of metoclopramide.

TABLE III
CONTRAINDICATIONS FOR METOCLOPRAMIDE

1. Absolute
 a. Pheochromocytoma
 b. Sensitivity or allergy or hypersensitivity
 c. Intestinal obstruction
 d. Intestinal perforation
2. Relative
 a. Extrapyramidal dystonias
 Parkinson's disease
 b. Sedatives
 Additive potential
 c. Anticholinergics
 Block metoclopramide

Summary

Metoclopramide is a procainamide analog which acts pharmacologically as a dopamine antagonist. It has been available in many countries in the world for the past 12-15 years. The most common usage is for nausea and vomiting. Controlled clinical trials in the United States have shown it to be effective for some patients with gastroesophageal reflux, and for most patients with slow gastric emptying. It reduces the time necessary to place intestinal tubes near the ligament of Treitz and may enhance the placement of upper intestinal feeding or decompression tubes. Since many sick patients have relative or temporary slowness in gastric emptying, it may prove effective in relieving the nausea and vomiting in these clinical situations. The major side effects are drowsiness, agitation, and extrapyramidal dystonic reactions. It is dangerous for patients with pheochromocytoma. It may have some additive effects if given with sedatives. Since most metoclopramide is excreted by the kidneys, a smaller dose may be required for patients with anuria or reduced renal function.

Self Assessment Questions

1. Metoclopramide is currently available in the United States for intravenous administration. In most other countries it has been used orally for years (and may someday be similarly available here) as a very effective and safe drug for:
 a. Suppressing Parkinsonian movements
 b. Controlling cardiac arrhythmias
 c. Short term local anesthesia and pain control
 d. Suppression of nausea and vomiting

e. Reversing intractable constipation due to colonic atony

2. Metoclopramide acts as a dopamine antagonist in the central nervous system. It therefore may cause:
 a. Extrapyramidal (Parkinsonian) movements
 b. Suppression of vomiting
 c. Prolactin release with galactorrhea
 d. Accelerated gastric emptying
 e. All of the above
3. Because of its primary route of elimination, the dose of metoclopramide should be reduced in patients with impaired function of:
 a. Liver
 b. Lungs
 c. Kidneys
 d. Biliary tree
 e. All of the above
4. In using metoclopramide to assist placing a duodenal tube, the dose may have to be reduced because of:
 a. Increased nausea and vomiting
 b. Cardiac effects
 c. Excessive drowsiness
 d. Anesthesia of mouth with numbness
 e. None of the above
5. Metoclopramide is contraindicated for patients who have:
 a. An allergic reaction with the drug
 b. Pheochromocytoma
 c. Organic intestinal obstruction
 d. Intestinal perforation
 e. All of the above

(Answers on page 424)

Malaria

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wishing the information to minimize the burden to those offering the information. Multiple forms were reviewed, designed, and modified to reach a reasonable compromise between convenience and burden of completion. The birth certificates of Kansas are unique with regard to information requested. This form provides statisticians with extensive data, and it has been utilized in several other states and statistical studies.

As of spring 1979, Wesley Medical Center with the assistance of UKSM-Wichita Statistics Division began the formal task of collating the data generated since 1974. Analysis of these data will provide needed information for optimizing educational outreach and patient care. One of the initial evaluations will be to compare outcomes of intrauterine transports with extrauterine transports. It is anticipated that this information will amplify the data from Milwaukee County General Hospital's 12-year review[6] and substantiate the findings of Pollitzer and Reynolds.[7]

Summary

The decrease in infant mortality since 1974 shows that progress is being made. Continued support of this program from the physicians of Kansas and the tertiary care centers, and the increasing number of secondary care centers is extremely important.

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The President's Message

Thank heaven this summer is over! Not only has it been hot, but the slow pace of KMS activities has been agonizing for me. With nearly a quarter of the KMS year gone we are only now getting underway with the very important business at hand — committee work.

When I was in fourth grade, a teacher had our class pause to watch the big wall clock as a minute ticked off. After the red second hand had made its full sixty-second sweep, she told us that minute was gone from our lives . . . forever. That taught me as a small boy to always try to use my time wisely, for as minutes pass into hours and hours pass into days, months and years, that time can never be recaptured. So it is that we must not fritter away the remainder of the year that we have to work together.

Lest I leave you with the impression that the summer has been wasted, let me assure you that I have completed what I consider to be the most important task of the president — the assignment of committees. We have highly qualified committee chairmen carefully selected for each of the committees, and over two hundred capable and interested physicians and spouses appointed to these committees. Each committee has been charged with several specific goals, and a member of our office staff has been assigned to each committee to help expedite the realization of those goals.



So the success of the KMS is now in the hands of your committees. It's time for us to take off and run with it for time is passing quickly.

Fraternally,



President



Informed Consensus

The human mind is attracted to increments of time as if the biologic clock gives out special beeps at certain times denoting significant segments. The annual event excites little but relief that the year is past and promotes excessive celebration in honor of this relief and a scarcely justified optimism for the future. A decade is more satisfying — always some failures and disappointments but a few successes that can be magnified by memory. The main interest, however, is to put a descriptive label on the decade which is presumed to recall the essence of the period. A century is too long for much personal identification — of interest primarily to serious historians, promoters of nostalgia gadgetry, and the oldest fogeys.

But fifty years is a particularly satisfying period for contemplation. With increasing life expectancy, there are more of us around to ponder the joys that weren't recognized at the time, the sorrows that bound us together, all the events that never really occurred (at least not as we remember them), the unrealized expectations, and the unexpected realizations. And as we become a nation of four living generations rather than three, we can join the weatherman and economists in searching for cyclic patterns to help us anticipate the future. It is a respectable period of time, sufficient to prompt someone to pronounce that we must learn from history's mistakes which only obscures the fact that history is an encyclopedia of questions, not answers. Fifty years is long enough to justify the propriety of age but short enough to command the awareness of youth. It encompasses problems that youth considers acute (and it will shortly solve) and age knows are chronic with the solutions being preamble to other problems.

All of which, beside indicating that a little summertime contemplation can be as dangerous as a little learning, serves to recall the 30s. One of the popular songs of the day was "Happy Days Are Here Again," a classical example of wishful thinking since the general state was one of apprehension. There was the real distress and social upheaval of a world-wide economic depression. Then there was what was known as the "Red Menace," a general certainty that Russia harbored a horde of booted and bearded Bolsheviks carrying sputtering bombs and about to spread out and envelop the world. The political ferment of the day produced a variety of "isms" whose distinctions were not too clear in the public mind except that they were not "capitalism" (which wasn't too well understood either) — in other words, they were foreign.

The situation brought about a polarization between those seeking to revitalize the old system and those who sought to replace it with a different one. A panoply of social changes was instituted, hailed by some, damned by others, and designated a great new deal or the tragic end depending upon which pole one frequented. Which ones were right and which were wrong was never satisfactorily determined since World War II came along to upset the protocol of the experiment and, like most wars (and time itself), it changed some problems, took the public mind off of others, and introduced a whole passel of new ones. (Any similarity to the current scene is worse than coincidental.)

The medical profession's favorite bugaboo at the time was called "socialized medicine" which called for numerous changes in medical practice, all of them anathema to the majority of practitioners of the day, particularly the idea of working for and being paid by the government. In retrospect, it is surprising that the changes in the medical area were not greater and earlier — perhaps in addition to the distractions of war, some of the significant achievements of the profession diminished any public wish to change the

medical system too much (although the presence of some very real successes in recent years hasn't diminished the call for changes today). Even so, it has been pointed out that most of the "radical" solutions advanced in those days (and successfully resisted, so the medical profession thought) have since become reality in one guise or another.

One still hears warnings of "socialized medicine," but the term seems outdated and inadequate for today's situation not only because of the extent to which its methods are now fact (even defended by the establishment) but also because there are some new features. Perhaps "publicized medicine" would be more appropriate, acknowledging the current surfeit of public involvement as well as the connotation of publicity (of which we seem to be getting lots). "Bureaucratized medicine" would be accurate but awkward. "Nationalized medicine" would please the promoters of national health insurance. We can't imagine its meeting with general approval but we offer as the threat of the 80s "consensualized medicine" which anticipates the increasing practice of determining proper medical behavior by consensus.

The pathogenesis of this condition would go something like this. Medical care costs have led increasingly to demands by those footing the bill — the government, insurance companies and other third parties, even second parties, the overlooked minority of patients who pay their own bills — for control of medical practice as a means of limiting the costs. Confronted as we frequently are by varying opinions in the handling of medical matters, it has become popular to call together experienced and knowledgeable professionals to come up with the one approved course of action. Applying the idea generally, diagnostic criteria and procedures can be prescribed by committee action (and limited, of course, to those procedures that give positive results since it has been established that anything giving a negative answer is, ipso facto, a waste of time and money, having the primary purpose of enriching physicians). With approved diagnosis established, therapy, also determined for each condition by consensus, will follow the accepted course. In extended form, this system can be computerized for rapid application and to simplify scrutiny by the committee to identify any nonconforming miscreants.

The consensus philosophy is basically of value and with a slight shift of perspective can be said to be the operating system we adhere to now. Confronted daily with the perplexities and complexities of human behavior under pathologic conditions, the physician's whole intent has been to select the one best approach to the diagnosis and the one most effective course of treatment for that patient. This has always been the motivating philosophy of legitimate medical function. So we're talking about the same thing? Well, yes and no. Yes if it is only a matter of manipulative semantics, but no if one approaches it from the standpoint of the vagaries of patient form and response and refusal to behave like an automaton. Without too much effort we can see this as another example of the way in which a system intended to benefit all patients would be incompatible with the care of a single patient who didn't know he was supposed to conform to the consensus.

There are two aspects of the matter that history — and recent history at that — can bring to mind. First, the demand for patient involvement (read public) in medical matters, whatever the virtues, would shortly result in the demand for inclusion of lay persons in those groups establishing the consensus. It may be quite proper by the rules of the new order to include nonprofessionals in such decisions, but the very least that would happen would be a dilution or distortion of the medical expertise going into the decision. We frankly doubt that this denigration of the physician will come to pass because we agree with the politicians who extol the wisdom of the public (at least at campaign time) — we think the people would not buy it if they can see how it will work.

The other factor, however, is the potent one. The effective pressure which would bring about such a system would be financial. The logical step, in the minds of the planners, would be to assign a dollar value to each course established by consensus, and financial control of medical costs would automatically be established. (Nor should one forget the impact that such a consensus determination would have on the liability picture since any departure from the accepted would be obvious. We assume, of course, that whatever changes the planners do bring about, they won't eliminate the malpractice factor.)

Extending the consensus idea to its extreme, then, we have a totally controlled plan in which the physician will need only a rudimentary knowledge of medicine, enough to get the patient programmed into the right channel where CCCC (Computerized Consensus and Cost Control) takes over and carries the patient through to conclusion. No, we don't really think it will happen — a war or some other social diversion will intervene. But the ingredients are at hand.

Well, it's been a long, hot summer. Makes you think of the 30s. — *D.E.G.*

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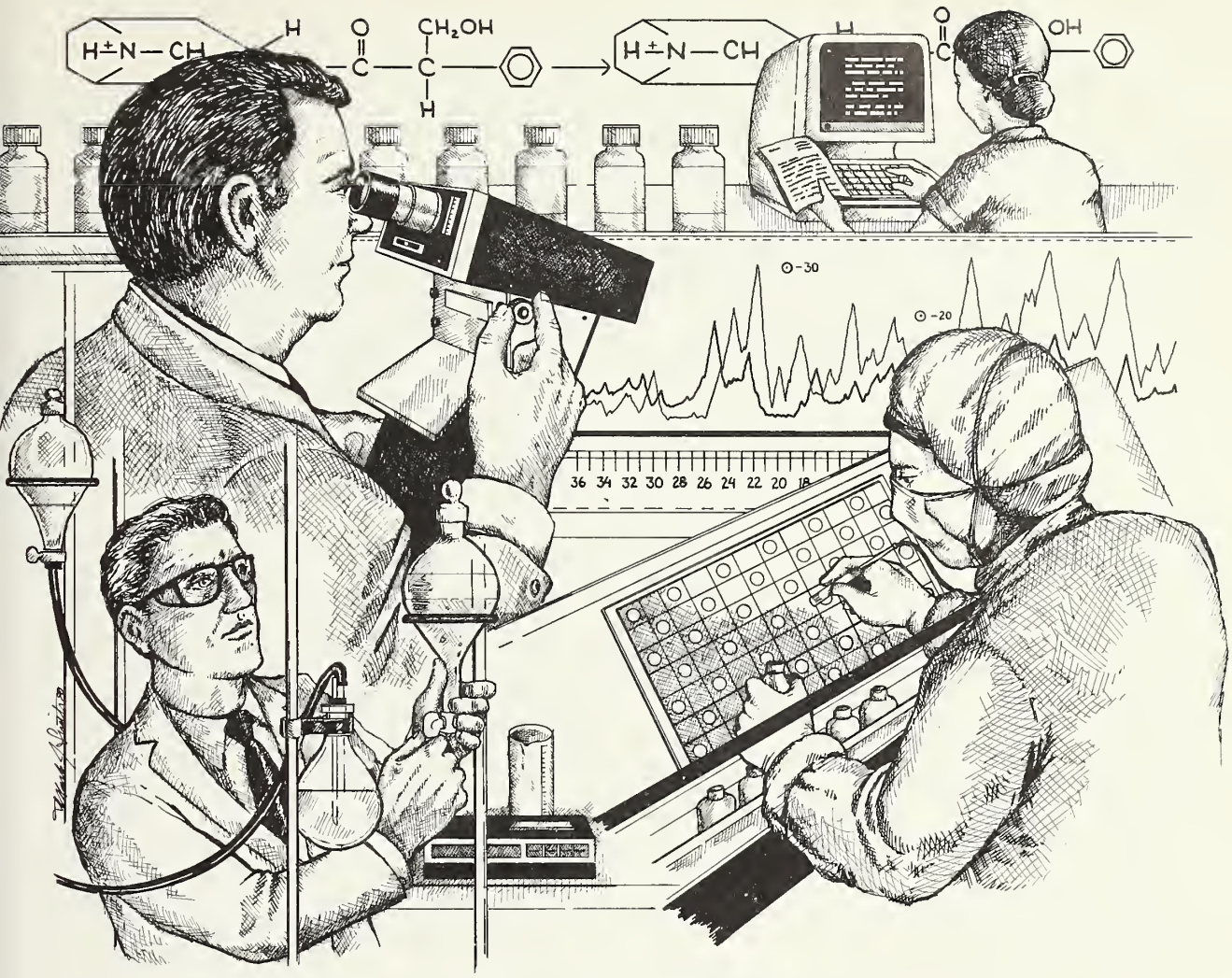
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Metoclopramide

(Continued from page 416)

Answers

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Indications and Usage: Symptomatic relief of anxiety, tension, agitation, irritability a insomnia associated with anxiety neuroses and transient situational disturbances; anxi associated with depressive symptoms and as a treatment of symptoms of anxiety if such syn toms are a significant feature of functional or organic disorders, particularly gastrointestinal cardiovascular

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by syste atic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CN acting drugs, warn patients on lorazepam not to operate machinery or motor vehicles, and diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with bar turates and alcohol have occurred following abrupt discontinuance of benzodiazepin (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Add tion-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillan when on benzodiazepines because of their predisposition to habituation and dependen Withdrawal symptoms have also been reported following abrupt discontinuance of benzo azepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid ov sedation.

Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result symptoms like those being treated, anxiety, agitation, irritability, tension, insomnia and occ sional convulsions.

Observe usual precautions with impaired renal or hepatic function.

Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepa has not been shown of significant benefit in treating gastrointestinal or cardiovascular comp nent.

Esophageal dilation occurred in rats treated with lorazepam for more than 1 year 6mg/kg/day. No effect dose was 1.25mg/kg/day (approximately 6 times the maximum huma therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn with 2 months of first observation. Clinical significance is unknown; but use of lorazepam for pr longed periods and in geriatric patients requires caution and frequent monitoring for symptom of upper G.I. disease.

Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia, some have h elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function te are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depress effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbi Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschi malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship dosage. Although all these anomalies were not present in the concurrent control group, th have been reported to occur randomly in historical controls. At 40mg/kg and higher, there w evidence of fetal resorption and increased fetal loss in rabbits which was not seen at low doses. Clinical significance of these findings is not known. However, increased risk of conge tal malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam a meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this per should almost always be avoided. Possibility that a woman of child-bearing potential may pregnant at institution of therapy should be considered. Advise patients if they become pre nant to communicate with their physician about desirability of discontinuing the drug.

In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepa and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like oth benzodiazepines. As a general rule, nursing should not be undertaken while on a drug sim many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and genera disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxio patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9% weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, na sea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, e function disturbance, various gastrointestinal symptoms and autonomic manifestations. In dence of sedation and unsteadiness increased with age. Small decreases in blood pressu have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind that multiple agen may have been taken. Manifestations of overdosage include somnolence, confusion and com Induce vomiting and/or undertake gastric lavage followed by general supportive care, monito ing of vital signs and close observation. Hypotension, though unlikely, usually may be controlle with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

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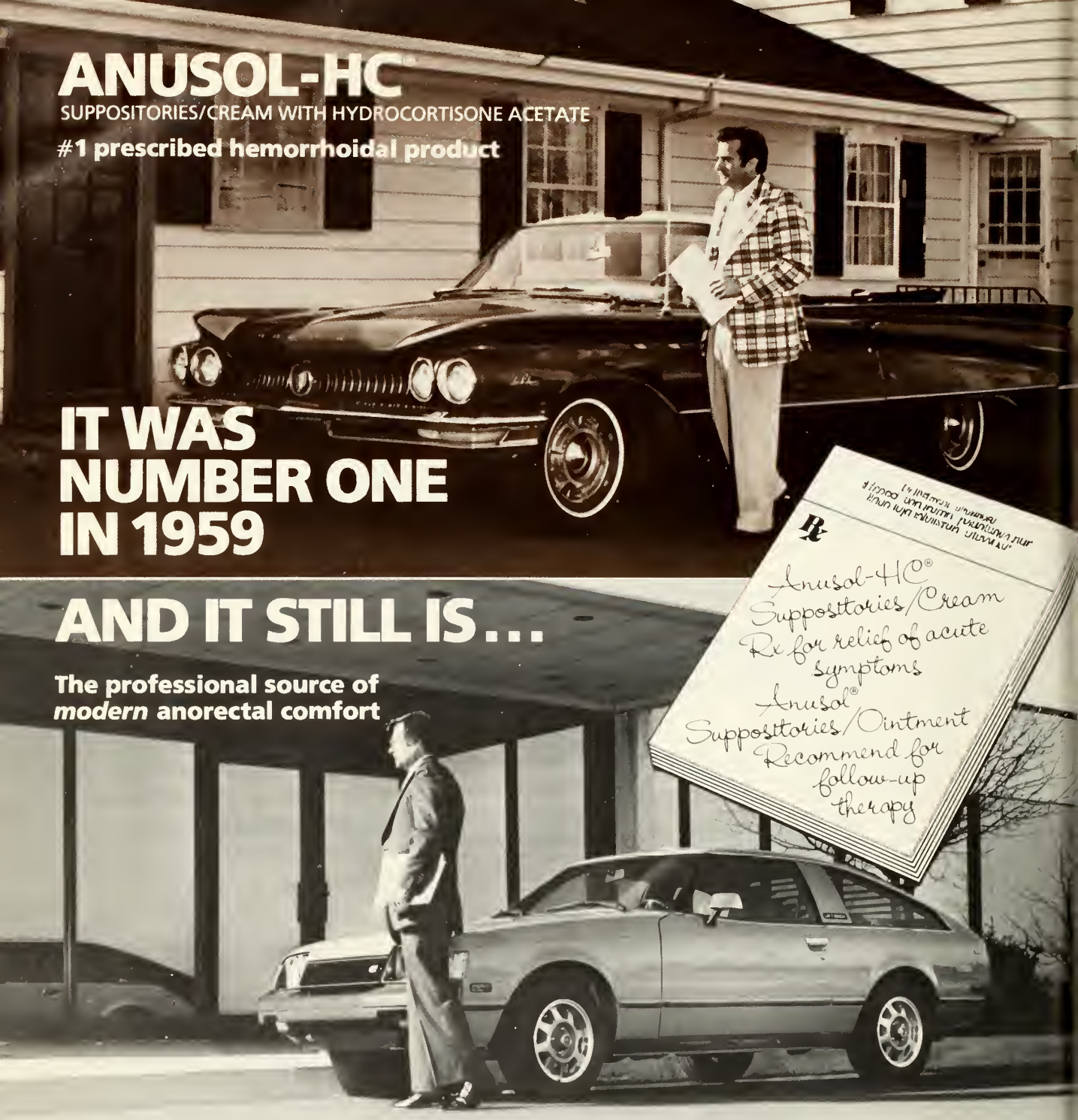
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Rectal Cream with Hydrocortisone Acetate
Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg, bismuth subgallate, 2.25%, bismuth resorcin compound, 1.75%, benzyl benzoate, 1.2%, Peruvian balsam, 1.8%, zinc oxide, 11.0%, also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg, bismuth subgallate, 22.5 mg, bismuth resorcin compound, 17.5 mg, benzyl benzoate, 12.0 mg, Peruvian balsam, 18.0 mg, zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

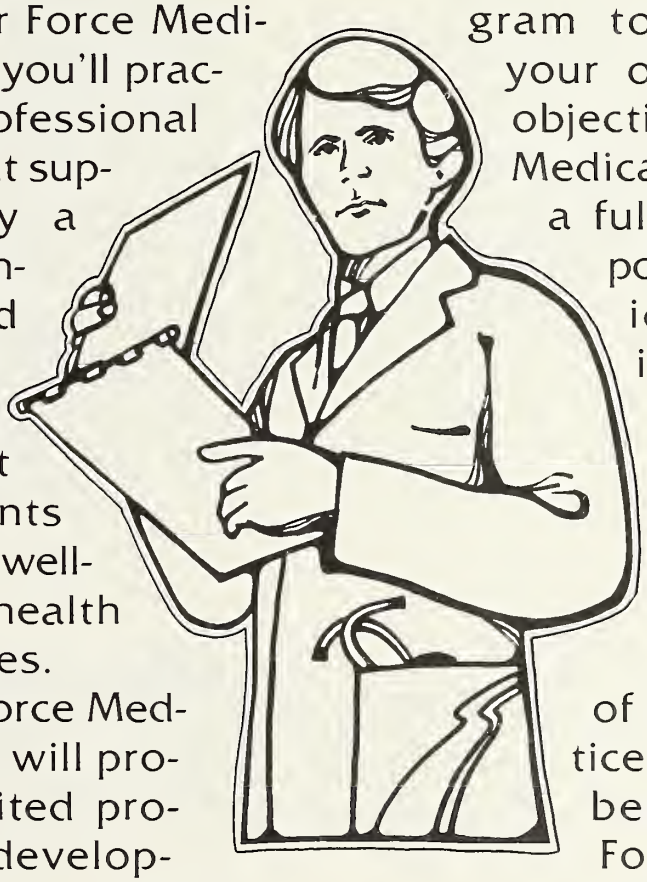
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Full information is available on request.

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Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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Product Information as of September, 1977

U.S. Patent 2,985,558

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Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

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Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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THE Journal OF THE Kansas Medical Society

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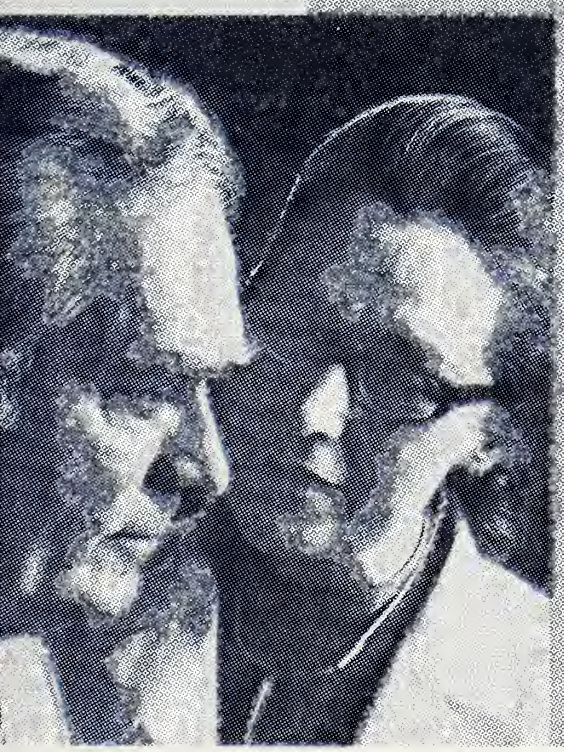
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Contraindications: Patients with known hypersensitivity to the drug

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema; minor menstrual irregularities; nausea and constipation; extrapyramidal symptoms; increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis); jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults.* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients.* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE Roche Products Inc.
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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.



A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC® (IV)

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.
Final classification of the less-than-effective indications requires further investigation.
The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor trenquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk es compared to maternel plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported; most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

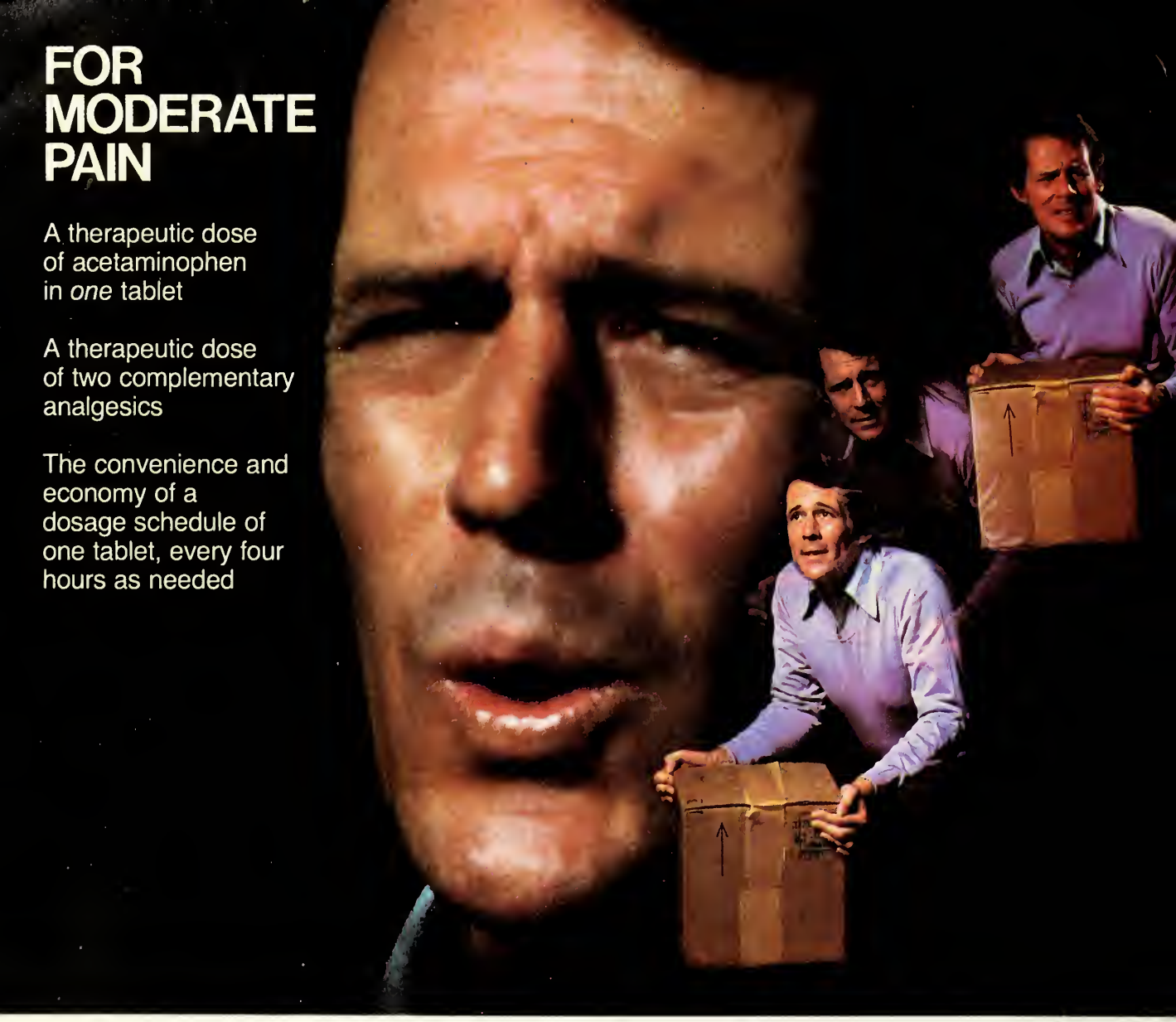
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FOR MODERATE PAIN

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WHY NOT WYGESIC® C IV

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE. Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY. Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS. The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (e.decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill, however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphen, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered, preferably IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Aneleptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information. (JAMA 237 2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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the great american smokeout
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The Great American Smokeout is a special day for cigarette smokers. It is a day on which they show themselves that quitting the habit is possible and not as difficult as many might fear. They do this by giving up cigarettes for 24 hours. Lots of them end up quitting for good.

The Great American Smokeout in Kansas will be more spectacular in 1980. Coach Don Fambrough of the Univeristy of Kansas and Coach Jack Hartman of Kansas State University have agreed to serve as the 1980 Honorary Smokeout Chairmen. They will be urging all Kansas smokers to kick the habit on November 20th.

The Kansas Division of the American Cancer Society will offer a poster and essay project for all grade levels. Participants will receive a Smokeout certificate signed by Coaches Fambrough and Hartman. The poster and essay project will encourage students to learn the positive health aspects of not smoking. Guidelines may be ordered by contacting the local American Cancer Society volunteers or the Kansas Division office.

Last year, the Gallup Organization reported that nearly 15 million smokers tried to quit on Smokeout Day and 5 million actually succeeded. In Kansas over 60 counties were involved in the Smokeout and this year we expect an even higher participation.

The media took up the Great American Smokeout and it received excellent coverage both nationally and statewide. In 1980 local volunteers will again be distributing TV and radio spots and press releases for the Smokeout.

Today, more than 100,000 Americans have lung cancer and smoking is responsible for close to 80 percent of those cases. The Great American Smokeout is now in its fourth consecutive year as a nation-wide event. The annual observance focuses public attention on cigarette smokers from coast to coast. The Smokeout is an up-beat, good-natured effort to encourage smokers to give up cigarettes for 24 hours, if only to prove to themselves they can.

We hope you will join the Great American Smokeout on November 20th. For more information call the Kansas Division of the American Cancer Society (913-267-0131) or write 3003 Van Buren, Topeka, Ks. 66611.



the great american smokeout
the great american smokeout
the great american smokeout



American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

AUXILIARY NEWS

An Open Letter to Kansas Physicians

Our KMSA President, Evelyn Huff, has asked that I write to you. Your KMS Committee for the Impaired Physician has been working to help physicians whose ability to give optimum care to their patients has become or may soon become impaired. As the physician's spouse may be the first to recognize early signs of trouble, your Committee decided to include the Auxiliary in its effort to reach impaired and potentially impaired physicians.

During the past years we have educated members of the Auxiliary about early signs of dependency on alcohol and other drugs, and touched generally on personality changes that might be indicative of physical or emotional disorder.

Aware that the physician's spouse may need help in coping with the physician's disease, we have made available, through contact with the KMS office, information about treatment centers throughout the state, Alcoholics Anonymous, Al-Anon and Al-Ateen, and other support groups. We also have available qualified physician's spouses to serve as support to families of impaired physicians. Help can be obtained with anonymity if desired.

We will, through this same medium, offer information to any physician's spouse who has a personal problem with chemical dependency or emotional distress. It is very difficult for the spouse (especially a wife) to seek help from the local medical profession. For this reason s/he may wish to seek help from someone in another area. The spouse's confidentiality should be protected as carefully as that of any other patient.

According to research done by C. B. Thomas and reported in the *Johns Hopkins Medical Journal,* of those physicians-in-training married during medical school, 50 per cent are divorced by completion of residency. Early lifestyles play a primary role in the development of lifestyles that will endure into the middle and later years. The KMS Committee and the Auxiliary have provided programs on the subject of medical marriage and danger of impairment for medical students, residents, and spouses whenever and wherever requested.

Our young physicians are especially vulnerable to impairment due to some of the prevailing psychosocial responses to stress. The use of mood-altering drugs and self-destructive measures may lead to early impairment while the physician is still in residency or even medical school. The well-being of the physician throughout years of practice may largely depend upon how well the physician-in-training succeeds in his/her professional and personal development.

Residency is a time of personal assessment and setting of priorities. If married, the resident and spouse need to work through these together so that both understand the demands of the profession and their own personal requirements. The demands of residency leave little time for building marriage relationships. However, neglect of the marriage and the resulting deterioration can lead not only to its breakup but to use and abuse of drugs by the resident and/or the spouse. The long range effect on spouse and children should not be underestimated. In visits with grown children of physicians, one frequently hears about the remote and busy father and involved mother who lacked a loving relationship. This situation is not unique to medical families, but as medical families are highly visible in their communities, their image has a significant impact.

There is need for appropriate professional and personal models for physicians-in-training, at the medical school and residency levels, who will provide guidance and set examples for neophyte physicians. The academic physician should be not only a

Mrs. Burnett served as President of KMS Auxiliary during 1977-78.

classroom instructor, but also mentor and counselor.

The young physician and spouse need social contacts with older physicians as well as peers and should be encouraged to continue spiritual and emotional development at a time when they need it most.

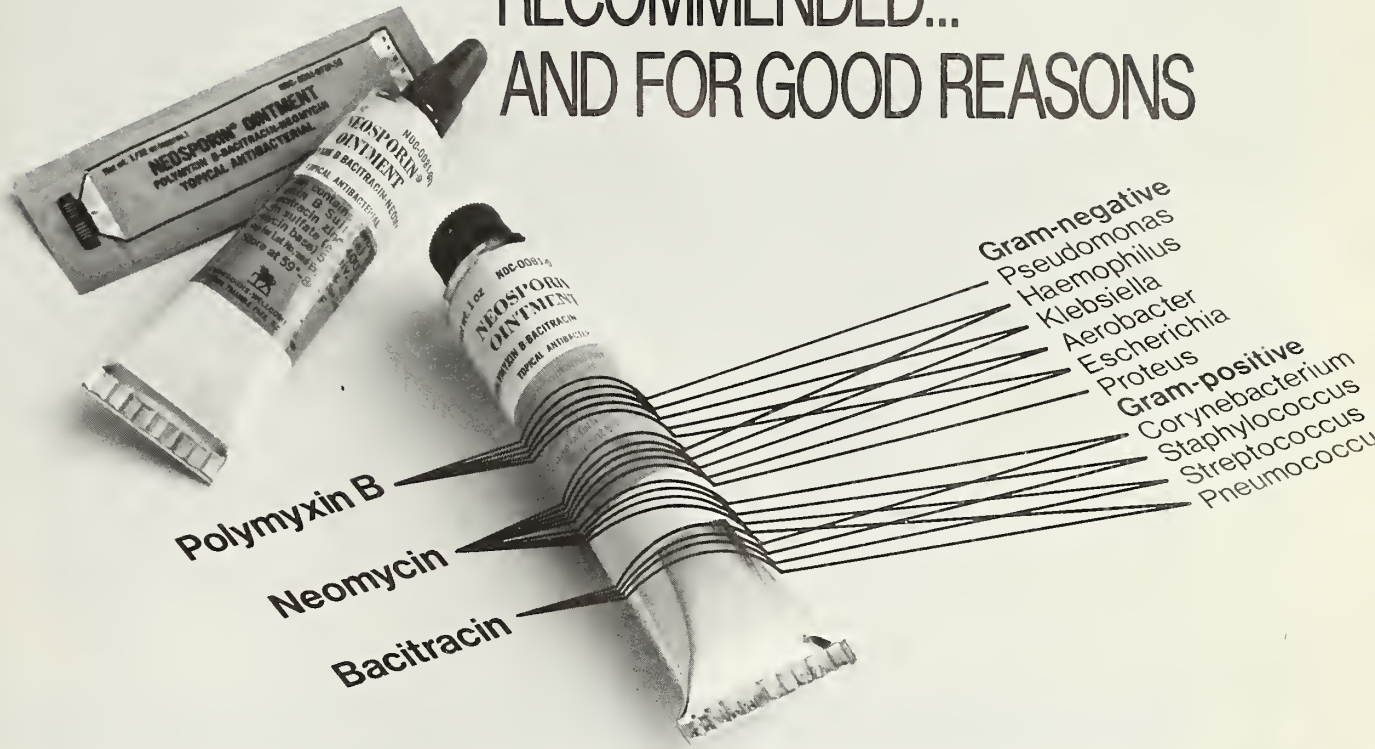
While organized medicine is endeavoring to assist directors of residency programs to meet the challenge of impairment and to promote well-being through medical education, we (both physicians and spouses) who are established in practice can contribute to helping young medical families to adjust to their new lives in "the real world." Sharing our friendship will mean much to them — and to us.

Perhaps an important element in prevention of impairment for both physician and spouse is interaction and friendship with others in the same profession, early and continuously throughout professional and personal development. The Auxiliary can help to facilitate such interaction. Auxiliary membership has helped me to understand the broader scope of marriage to medicine in ways that being a nurse could not. I found understanding friends who share my admiration of the profession and my frustrations with its demands. We also share the bond of mutual endeavors to work in and for our communities, helping to bring healthier lifestyles to all.

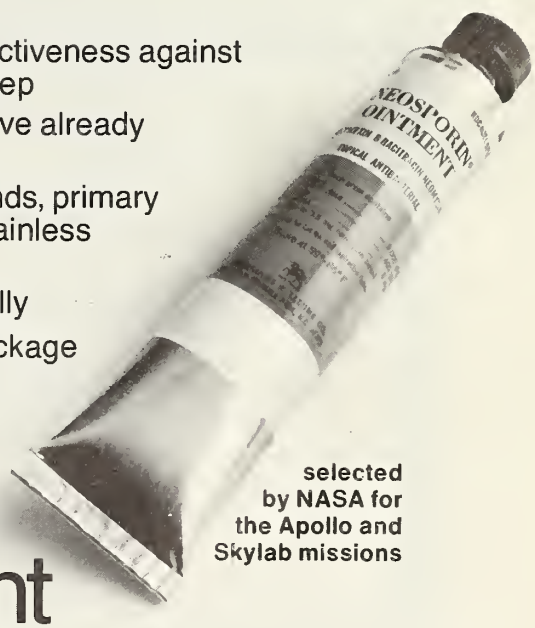
Sincerely,
Jackie Burnett, Liaison
KMS Committee for the Impaired Physician



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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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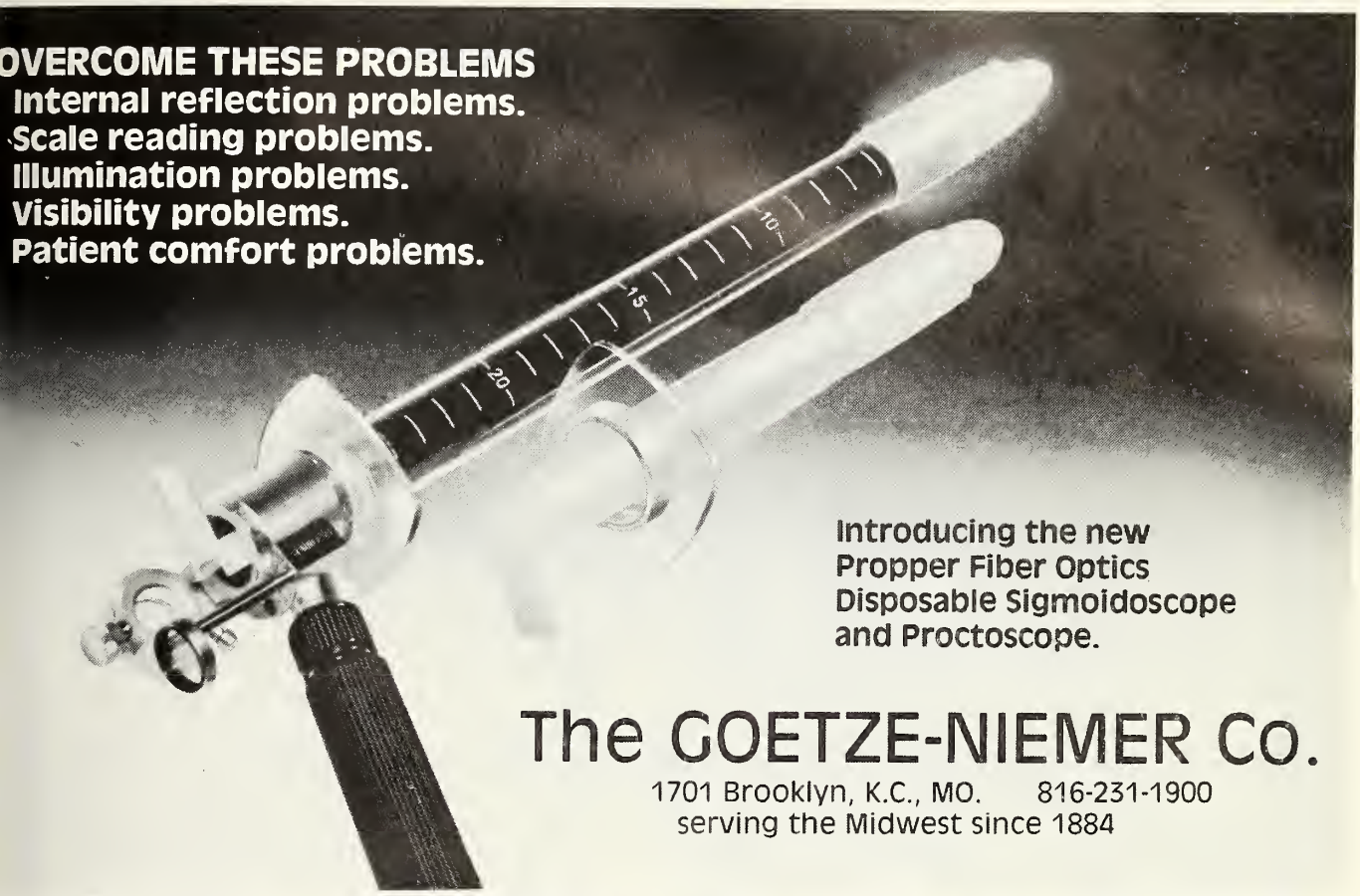
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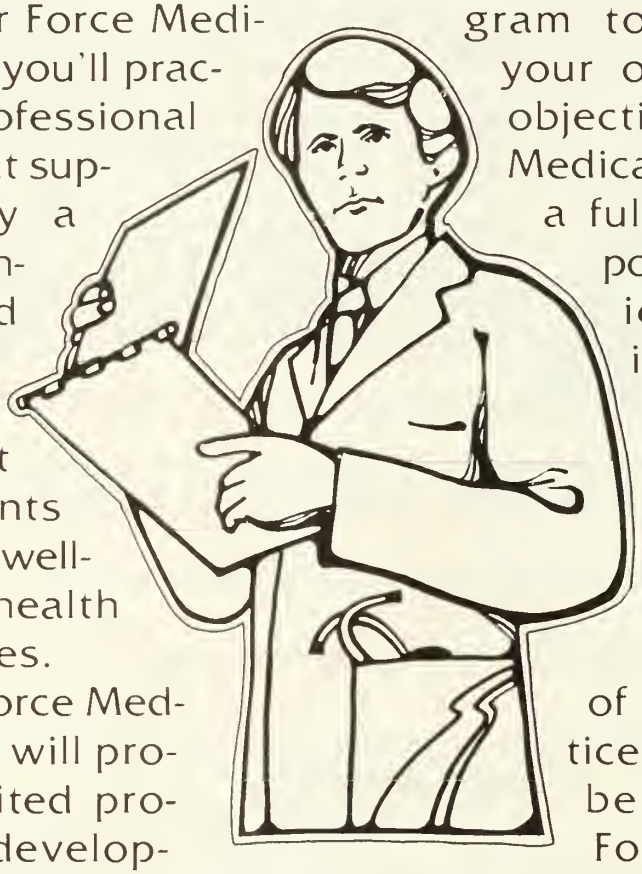
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AMA Interim House of Delegates Meeting

December 7-10, 1980 San Francisco Hilton

Members are encouraged to attend and help guide the reference committees' deliberations. Clair Conard of Dodge City will chair Reference Committee H.

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The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

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Medical HISTORY

The Smallpox Story

Man Finally Defeats an Old Adversary

ABBAS M. BEHBEHANI, Ph.D.,* *Kansas City, Kansas*

Smallpox, a disease of antiquity, has killed innumerable millions of people. Variolation — immunization with material from smallpox lesions — was practiced in the Far East for many centuries and was introduced into Britain in 1721. Jenner introduced the safer vaccination process with cowpox virus in 1796. Incidence of smallpox then declined steadily in Europe and North America, but prevailed elsewhere until the World Health Organization (WHO) initiated a global eradication program in 1967. Subsequently the last case of naturally occurring smallpox recovered in Somalia in October 1977. There is current concern about the possible emergence of naturally existing monkeypox and whitepox viruses as agents of human poxvirus disease.

Early Period

DESCRIPTION OF SMALLPOX appears in the earliest Egyptian and Chinese writings. The mummy of Ramses V, who reportedly died of an acute illness at the age of 40 years in 1100 B.C., shows numerous pustular lesions on the face that closely resemble those of smallpox. The Persian physician Rhazes (860-932 A.D.) wrote the first differential and graphic description of symptoms of smallpox and measles and stated that smallpox was described by Galen in the second century. However, it is generally believed that, although smallpox was prevalent in China and India some 1000 years before the advent of Christianity, the first recorded epidemic of this disease further west occurred in Arabia during the sixth century. The disease was then disseminated to North Africa and Europe by the Arab invaders. Early in the sixteenth century, smallpox appeared in Britian while the slave ships from Africa spread the disease to the West Indies and Central America and subsequently the Mexicans suffered heavily from this scourge. (It is believed that 3½ million Indians died of smallpox in the ensuing epidemic.) During the seventeenth and eighteenth centries, smallpox caused deadly endemics and epidemics in Britain; at times and in certain cities, the smallpox mortality was not less than one-sixth of the birth rate. In the United States, smallpox epidemics, involving occasionally one third of the population, occurred frequently during the eighteenth century, and in contrast to Britain where 90 per cent of cases occurred in children younger than ten years, Americans of all ages were afflicted. For example during a smallpox epidemic in Boston in 1721, 5,759 cases with 842 deaths were recorded among 10,700 citizens of that city.[1]

Introduction of Variolation Into England

The ancient Chinese initiated immunization against this disease by variolation. According to Francois M.A. Voltaire, dried powder of smallpox crusts was inhaled through the nose in a manner similar to taking the snuff. In the Near East (*e.g.*

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Greece, Turkey, and Arabia), a modified method of variolation — namely, removing some of the thick liquid from a smallpox pustule and rubbing it into a small scratch made with a needle on the arm of a child — was widely practiced. The Circassians (inhabitants of a region north of the Caucasus Mountains on the shores of the Black Sea) practiced variolation as a preventive measure against death and disfiguration of their daughters from smallpox. These people were poor and sold their beautiful daughters to the Sultans of the Ottoman Empire and the Shahs of Persia.

Around the turn of the seventeenth century, a considerable number of the British (as well as the French, Italians, and Germans), including reputable physicians, became aware of variolation, as practiced outside of Europe, for protection against smallpox. In late 1712 and early 1713, Richard Waller, Secretary of the Royal Society of London, started a campaign to better inform Society members of the practice of variolation by soliciting correspondents in foreign countries and the British colonies. Subsequently, on May 27, 1714, Dr. John Woodward communicated a letter in Latin from Dr. Emanuel Timoni, dated Constantinople, December 1713, to the Royal Society. A translation of this letter was read to the Society on June 3 and was discussed by the members on June 10, 1714. Dr. Timoni (of Italian birth) had a medical degree from Padua and another degree from Oxford, and was a member of the Royal Society. His letter described the art of variolation, as observed and practiced by him, in great detail. It was published in the Philosophical Transactions of the Royal Society for April, May, and June, 1714. However, more information and confirmation of Timoni's account were requested by Society members. Consequently, Secretary Waller wrote to Dr. William Sherard, the British Consul at Smyrna (now called Izmir in Turkey) for more information. In March 1716, Dr. Sherard sent to his brother James, a Fellow of the Royal Society, a letter with a printed pamphlet (published at Venice in 1715) by Dr. Jacob Pylarini, former Venetian Consul at Smyrna. This pamphlet confirmed Timoni's account of the practice of variolation and contained personal observations made while practicing in Smyrna and Constantinople. It was promptly printed in the Philosophical Transactions of January-March 1717. The above two communications, however, aroused little interest in Britain because they were regarded as virtuoso-amusements.

The awareness of the British people of the practice of variolation in foreign lands through traders, ambassadors, missionaries, sailors, and the two scientific communications mentioned above, apparently needed a third element for the introduction of variolation into Britain. This was effectively provided by the unconventional and forward Lady Mary Wortley Montagu who was the wife of the British Ambassador to the Ottoman Empire and lived in Adrianople and Constantinople during 1717-1718. She was stricken, as a young wife of 26 years of age, with smallpox in 1715 and suffered a pockmarked skin and loss of eyelashes. While in Turkey she became enthusiastically interested in the practice of variolation as a preventive measure against smallpox. In 1717, she wrote to her friend Sarah Chiswell of Nottingham "The smallpox so fatal and so general among us is here entirely harmless by the invention of ingrafting which is the term they give it." She described the operation "by scratching open a vein in the patient and putting into it as much of smallpox venom as could lie on the head of a needle." She further wrote that she was so much satisfied with the safety of variolation that she intended to try it on her dear little son and that she was patriotic enough to take pains to bring this useful invention into fashion in Britain.

On March 18, 1718, Lady Mary had her 6-year-old son, Edward, variolated (reportedly despite the opposition of the Embassy Chaplain who called variolation an un-Christian operation that could succeed only in the infidels).* Her infant daughter, however, was not variolated at that time because her nurse had not had smallpox. The immunization was successful and after her return to Britain, she informed her friend Caroline, the Princess of Wales (who later became the Queen of King George II) of her experience with this practice.

In the spring of 1721, a severe smallpox epidemic, involving both children and adults, broke out in Britain. In March an anonymous pamphlet on variolation was produced by a London physician (a Portuguese named à Castro), and there was an address on April 17 by the well-known Dr. Walter Harris before the Royal College of Physicians in which the practice of variolation in Constantinople was mentioned. Nevertheless, it was the private action of Lady Mary that gave the needed impetus to the start of variolation in Britain. In April 1721, she sent for Dr. Maitland (who had retired to the country)

* The variolation of the boy was most likely suggested or approved by Dr. Emanuel Timoni (who was engaged by Lady Mary's husband Edward as the family physician) and was performed with the supervision and participation of Dr. Charles Maitland, the Embassy surgeon. In contrast to his younger sister Mary (see text), Edward, Jr., grew up to become a rascal and a constant embarassment to his parents.

and asked him to variolate her 3-year-old daughter, Mary, the future Countess of Bute and wife of Conservative Prime Minister Lord Bute. The girl was variolated in both arms and when the pocks appeared, she was examined separately by three members of the College of Physicians. This successful operation was of signal importance as variolation had been hitherto considered as a virtuoso-amusement until Lady Mary sponsored it by having her own daughter variolated. Furthermore, Lady Mary gained support from her friend the Princess of Wales who requested that six condemned criminals be allowed to volunteer for variolation with freedom as their reward should they survive. Dr. Maitland variolated the prisoners; all survived and were released. A short time later seven orphan children were also successfully variolated. This was followed by the variolation of two of the Princess's daughters and Lord Bathurst's six children. However, following these variolations, one of Bathurst's servants and the young son of the Earl of Sunderland died following inoculation. Much publicity and some opposition by the clergy and the medical profession followed. However, many prominent London physicians (*e.g.* Sir Hans Sloane, Dr. James Jurin, Dr. John Arbuthnot, Dr. James Keith, and Dr. Richard Mead) gradually endorsed the practice. In defense of variolation, Lady Mary published an anonymous essay entitled "A Plain Account of the Inoculation of the Smallpox by a Turkey Merchant" in a popular London newspaper (*The Flying Post* or *Post-Master,* September 11-13, 1722). Subsequently, variolation became more popular and by the end of 1722, a total of 182 people had been variolated. Lady Mary became commonly regarded in Britain as the introducer and popularizer of variolation.*

The practice of variolation, however, did not become widely accepted in Britain as it entailed some risk to the inoculated individuals and their contacts. The vaccine, taken from the lesions of a smallpox case and introduced to the skin of the vaccinee by incision or puncture, produced a local lesion, a fever starting on the seventh or eighth day, and a general eruption on the ninth or tenth day. Although the inoculated smallpox had a shorter incubation period and was milder in most vaccinees than the natural smallpox, 17 of 897 variolated persons died during the first eight years of this practice. Moreover, the danger of variolated persons spreading the disease to their contacts was well recognized. Hence variolation fell into disfavor during the 1730s in Britain.

In Colonial America, the Reverend Cotton Mather of Boston, who had some knowledge of the practice of variolation in Africa through his Negro slave, read Timoni's letter in the Philosophical Transactions and in 1721, when an outbreak of smallpox occurred in Boston, actively urged Boston physicians to practice variolation. Initially only Dr. Zabdiel Boylston was persuaded and on June 26, 1721, he variolated his son Thomas and two slaves. Seven more persons were variolated by July 17. Dr. Boylston, who lacked the M.D., was promptly opposed by all his colleagues including Dr. William Douglass who was the only holder of the M.D. in Boston. However, six of the most prominent clergymen defended Dr. Boylston and the righteousness of his experiments. Dr. Boylston subsequently reported that he had variolated 242 individuals of whom six died for a mortality rate of 2.5 per cent as compared to 15 per cent among naturally occurring smallpox cases. However, in 1722, Boston authorities placed the practice of variolation under public control and Dr. Boylston promised not to variolate anyone without official approval.

Interest in the practice of variolation, however, was later revived in Britain due to a modification of the procedure introduced by Drs. Mowbray (a surgeon) and Kilpatrick (a physician) during the 1738 smallpox epidemic in Charleston, South Carolina. Mowbray used material from the pustules of a variolated person (instead of a smallpox case) for inoculation and repeated the process up to six times without any loss of infectivity. This modification reduced the risk of generalized disease significantly and lead to the resumption of variolation in Britain. Other Western European countries initiated variolation after 1750.

* Lady Mary (1689-1762), the daughter of Evelyn Pierrepont, the First Duke of Kingston, married Edward Wortley Montagu, M.P., and later Ambassador to Turkey, in 1712 after a long bickering courtship. Faced with a troublesome and unhappy marriage in the 1720s, she looked elsewhere for emotional satisfaction. As she was gifted with an active intellect and a wide-ranging curiosity, she quickly emerged as a prominent and widely admired letter-writer, strong advocate of variolation, moral and political reformer, and feminist. However her intense romantic attachment, at age 47 years, to the handsome and highly cultured but effeminate Venetian Count Francesco Algarotti (who was half her age) led to her retirement to Italy in 1739 where she lived, away from her husband and children, for more than 20 years (the two were together only in Turin for about two months in 1741). In 1762 she returned to London where she died at age 73 years from breast cancer. Lady Mary was an extraordinary woman who went beyond the boundaries of her time and class. In her feuds and eccentricities, she acquired a great number of admirers as well as many opponents; notable among the latter were poet Alexander Pope, and author Horace Walpole (Fourth Earl of Oxford and M.P.) who described her as a dissolute and profligate heartless woman.[7]

In this country, variolation was widely practiced both before and during the Revolutionary War. However, concern about the possibility of variolated individuals transmitting the disease to others caused several of the 13 colonies, at one time or another, to pass laws against variolation or at least against its practice outside of very strictly controlled variolation hospitals. Hence by 1776 a number of cities were officially anti-variolation. Consequently, it has been suggested that the reason for the severe outbreaks of smallpox among the colonial troops was that only a small percentage of the soldiers had been variolated.[1-8]*

Jenner Substitutes Vaccination For Variolation

Dr. Edward Jenner was born on May 17, 1749, in Berkeley near Bristol in Southern England. He was the youngest son of the vicar of Berkeley and lost both parents at the age of 5 years. His elder brother, who replaced his father as vicar, directed Jenner's early years and the family decided on a medical profession for the young lad. Following primary education at local schools, Jenner, at the age of 13 years — as was customary for medical education at that time — was apprenticed for seven years to a Sodbury surgeon named Daniel Ludlow. He then spent two years as the first resident student of the great surgeon and innovator of contemporary medicine, Dr. John Hunter, working at the newly established St. George's Hospital in London. During these two years, Jenner acquired much experience at the most up-to-date school of his time and began a lifelong friendship with the great innovator. In addition to his medical practice, Hunter was a great naturalist and thus the young Jenner, being a keen naturalist himself, followed the guidance of his mentor and worked enthusiastically for many years on such special studies as the habits of the cuckoo, body temperatures of hibernating hedgehogs, and bird migration; these studies resulted in several scientific publications.

At the completion of his residency at St. George's Hospital, Jenner was offered a lucrative medical practice in London but he opted to go back to Berkeley and spend the rest of his life — except for brief visits to Cheltenham and London — as a country physician. He even did not attempt to take the examination on Classics in order to become a member of the Royal College of Physicians. As a practicing physician in Berkeley, Jenner studied various disease processes enthusiastically and performed postmortem examinations. He described the endocardial results of acute rheumatism and formulated a mercurial ointment for ophthalmia later called the "Golden Eye Ointment." For his significant contributions to medicine, the University of St. Andrews in Scotland granted him the degree of Doctor of Medicine. During two serious illnesses — typhus and a severe frostbite — he recorded his own signs, symptoms, and progress with remarkable clarity and clinical detachment.

The notion of cowpox protecting against smallpox was presented to Jenner, while he was an apprentice to Dr. Ludlow in Sodbury, by a dairymaid who told him in 1770 that she could not catch smallpox (which was a ravaging disease at that time) because she had had cowpox. This notion was well established among farmers. It is reported that in the spring of 1774, a Dorsetshire farmer named Benjamin Jesty, who had had cowpox as a young man, vaccinated his wife Elizabeth and his two sons, Robert and Benjamin aged 3 and 2 years respectively, with material taken directly from the udder lesion of a cow with cowpox from his neighbor's herd. He used a "stocking needle" and inoculated his wife on the forearm (because of her dress) and his sons on the upper arm. However, his wife developed a septic infection in addition to cowpox and he was reproached for this by his neighbors. He did not inoculate any other person. Most probably, other laymen performed similar prophylactic procedures but all those inoculations involved using material obtained directly from cows. There is no evidence to indicate that Jenner was aware of these inoculations when he initiated his vaccination experiments.

The Sodbury dairymaid's remark about the protective effect of cowpox against smallpox apparently left an indelible impression on Jenner and more than 20 years later, when he was 40 years old, he started to investigate the validity of this country lore. Jenner himself was variolated as a boy in 1756 and nearly died of the combined effects of medication (drastic purgation and bleeding in preparation for variolation) and disease. Thus the search for a safe effective procedure against smallpox became of immense importance to him. For about ten years he carefully studied and recorded cases who became refractory to variolation after being infected with cowpox. This

* The severity of the smallpox outbreak among the Colonial troops is vividly illustrated by the following two statements from eyewitnesses. Governor Jonathan Trumbull of Connecticut, who visited the retreating American troops following the failure of the assault on Quebec in July 1775, stated, "I did not look into a tent or hut in which I did not find either a dead or dying man." A physician who attended these troops said, "I wept till I had no more power to weep." (See Carrington, Henry B., *Battles of the American Revolution, 1775-1781*, New York, Promontory Press, 1973.)

investigation convinced him of the protective effect of cowpox against smallpox. Consequently, he planned to use material from a typical human cowpox lesion as inoculum for immunizing susceptible humans and also to transfer cowpox from person to person without resorting to the bovine disease for inoculum material. On May 14, 1796, Jenner immunized 8-year-old James Phipps of Berkeley with material obtained from a typical cowpox lesion on the hand of a milkmaid named Sarah Nelmes who acquired it from a Gloucestershire cow called "Blossom." The immunization produced a relatively mild vesiculating lesion which healed within two weeks. The boy, who had never had smallpox, was challenged with material from a real case of smallpox on July 1, 1796; he showed a solid immunity. Jenner submitted the details of this experiment to the President of the Royal Society, Sir Joseph Banks, who was not impressed by the manuscript. However, Jenner was undaunted and repeated his procedure many times with successful results during the following two years. He used quills to store lymph from case to case and observed that the inoculum remained viable for days or even weeks. In June 1798, he published a 64-page monograph entitled "An Inquiry Into the Causes and Effects of the Variolae Vaccinae,* a Disease Discovered in Some of the Western Counties of England Particularly Gloucestershire, and Known by the Name of Cow Pox." The second and third editions, with added data, were published in 1800 and 1801. Jenner's proposal, which was based on conclusive evidence, met with the violent opposition of his medical colleagues. He spent three months in London trying in vain to convince his colleagues of the validity of his immunization procedure which had been characterized as unnatural and dangerous. Later he was confronted with cartoons showing vaccinated babies growing cowhorns, disreputable books ascribing strange results to vaccination and mere vicious abuse by certain antivaccination people. Moreover, a number of London physicians tried to take the title of "Discoverer of Vaccination" away from him and brought Benjamin Jesty and his son Robert in 1805 to London to testify in an attempt to prove that Jenner was a fraud. Our medical hero was immensely hurt by the deeds of this group whose motivation was solely a spiteful jealousy.* Nonetheless, Jenner continued his medical practice in Berkeley and vaccinated hundreds of poor people (at a summerhouse called "The Temple of Vaccinia") who came to him for this purpose. Meanwhile in London, surgeon Henry Cline (one of Hunter's old students) used some of Jenner's dried lymph (stored for three months in a quill) which was left with him by accident, as a counter irritant in a boy with an inflamed hip joint and was informed subsequently by Dr. Lister that his patient became immune to inoculated smallpox. This observation, publicized by Cline and Lister, gave Jenner's vaccination procedure its needed impetus and thus the practice of vaccination became popular in Britain. Elsewhere, *e.g.* France and Russia, the Jennerian vaccination became even more popular. Dr. Benjamin Waterhouse, who inoculated members of his household in 1800, is credited with the popularization of vaccination in the United States. In order to provide viable lymph to distant lands such as the United States and India, Jenner organized relays of vaccinated emigrant children. The Jennerian innovation was adopted by the whole world within ten years. Jenner received many honors and awards from emperors and kings and from various groups and organizations throughout the world. A wampum belt sent to him by the Nine Nations of the North American Indians was especially valued by Jenner and he wore it with pride on ceremonial occasions. Dr. Jenner died from a stroke on January 25, 1823.[9-10]†

Jenner's vaccine was maintained by arm-to-arm passage for many years; however, fresh material from an active case of bovine cowpox was added occasionally. In 1845, Negri of Naples propagated the vaccine virus in cows, initially using humanized lymph, and thereafter the bovine lymph from inoculated cows was used for human vaccination and propagation of virus in cows. This method of preparation spread to France in 1866 and subsequently to

* Variolae vaccinae, which means smallpox of the cow, was coined by Jenner and from it the term vaccination (or cowpoxing as it was called by some) was derived. Jenner, however, was bitterly attacked by one of his outspoken opponents — Dr. E. M. Creighton — for coining this Latin phrase because he (Jenner) had not taken the examination on classics.

* Jenner's opponents were a formidable group of prominent London physicians. They included Drs. C. Crookshank and E. M. Creighton who called Jenner a cunning charlatan. Drs. G. Pearson and W. Woodville accepted the Jennerian vaccination and practiced it widely but refused to acknowledge that Jenner was its innovator.

† In 1977 Dr. Peter Razzell wrote two books entitled "The Conquest of Smallpox" and "Edward Jenner's Cowpox Virus." In these two books the author argued that variolation was remarkably safe and effective and that smallpox would have been eliminated whether or not the Jennerian innovation had ever been introduced. He further states that Jenner's vaccine contained an attenuated smallpox virus and not cowpox virus. The British author accuses Jenner of deliberate distortion of evidence and self deception. This reviewer obviously does not agree with Dr. Razzell. Interested readers are referred to the two books published by Caliban Books, Sussex, England.

most of Europe. In the United States, calf lymph was used in 1870, the original lymph being obtained from France. The technique of scarifying the entire flank of cow and producing a large quantity of vaccine was eventually introduced. Early in this century in certain localities of India, an inoculated cow was led from door to door and a bit of matter was scraped off for vaccination at each door.

The vaccine virus is now called vaccinia which is, in its present form, different from both the original cowpox virus and the smallpox virus. It is believed to be a hybrid that arose from the inadvertent mixing of the two viruses during the early vaccination programs. However, the vaccinia virus is relatively avirulent for man and produces an effective immunity against smallpox. The vaccines last used in the United States, were mainly glycerinated lymph from infected skins of calves or sheep and infected chorioallantoic membranes of embryonated eggs. In addition, lyophilized lymph and cell culture grown vaccines were also available.

Smallpox After the Jennerian Innovation

Following the introduction of vaccination by Edward Jenner in western countries, the incidence of smallpox declined steadily and with the exception of a few epidemic years (about 49,000 cases and 173 deaths recorded in 1930 in the United States, and about 14,900 cases and 47 deaths in 1927 in Britian), this disease became of lesser importance as a cause of death among children than measles and scarlet fever. However, elimination of smallpox in Europe and North America did not occur until the 1940s with initiation of more widespread vaccination and intense efforts to stop outbreaks where they occurred. This delay has been attributed to the fortunate or unfortunate replacement of the virulent variola major virus strain (mortality rate up to 45%) by the much less virulent strain of variola minor virus (mortality rate about 1%) in Europe and the Americas around the turn of the century. The variola minor strain apparently originated in South Africa and spread only to the two above-mentioned continents; in the rest of the world, however, variola major virus remained the dominant strain. Thus elsewhere, where smallpox remained endemic, death from this disease prevailed; during 1950, a total of 157,322 cases with 41,092 deaths were recorded in India alone.[11]

In regard to the pathogenesis of this infection, as it occurred in recent years, smallpox has been classified as two related diseases, namely, variola major (classic or Asian smallpox) and variola minor (alastrim or African smallpox) caused by two viruses with distinctive characteristics. In contrast to variola minor which has a negligible case fatality rate, variola major has an overall case fatality rate of 15-45 per cent. The World Health Organization (WHO) recognizes six types of variola major with different case fatality rates: (1) ordinary-discrete, <10 per cent; (2) ordinary-semiconfluent, 25-50 per cent; (3) ordinary confluent, 50-75 per cent; (4) flat, >90 per cent; (5) hemorrhagic, almost 100 per cent; and (6) modified (altered by previous vaccination), <10 per cent.[12] The relationship between the clinical severity of the disease and the virulence of the causative variola virus strain has been established.[13]

The virus enters the susceptible host through the mucous membrane of the upper respiratory tract, and after local multiplication, is drained by the lymphatics to the regional lymph nodes. Here further multiplication occurs and the virus then enters the blood stream causing the primary viremia. This is followed by the invasion of the reticuloendothelial system and extensive multiplication of the virus leading to the secondary viremia. The virus then invades the epidermis causing the skin eruptions. Clinically, after an incubation period of about 12 days, the pre-eruptive symptoms of headache, fever, malaise, prostration, pain in the back and limbs, and vomiting may appear suddenly or gradually. The skin eruptions usually develop after three to five days; they appear as one crop of macules which successively change to papules, vesicles, and pustules. The pustules start to scab toward the end of the second week and the crusts fall off in about a week leaving a pink scar which gradually fades in color (pock marks). The distribution of the skin lesions is typically centrifugal showing the greatest concentration on the face, forearms, wrists, palms, and soles as well as in the mouth and throat. The chest, abdomen, thighs, and the upper arms are relatively spared. The skin eruptions coincide with a fall in temperature (presumably due to the appearance of antibodies) which is followed by a rise in temperature (presumably due to absorption of toxic products of necrotic cells) and the pustulation of the vesicles. Conjunctivitis is manifested in some patients during the first eight days of illness. Bacteria, especially staphylococci, may contaminate the pustules leading to a variety of complications such as abscesses, septic joints, osteomyelitis, and corneal ulcers leading to blindness. The cause of death is not well-understood; general toxemia, septic shock, and disseminated intravascular coagulation (in hemorrhagic type) have been suggested.

Mild or modified variola major may occur in partially immune individuals or following infection with a less virulent strain; such cases may be mis-

taken for variola minor. The latter disease (also called alastrim) has a pathogenesis similar to that of variola major but the clinical features are much milder. The skin lesions generally are fewer, smaller, and more superficial; they remain discrete and evolve quickly. The general condition of the patient is usually good and convalescence is rapid. Bacterial contamination and complications are very rare.

Histopathologically the skin lesions start with the proliferation of the prickle cells due to viral invasion. Dilation of capillaries in the corium with swelling of the endothelial lining and infiltration of mononuclear cells — especially around the vessels — are also present. The malpighian cells then become edematous and undergo ballooning degeneration. The cell walls of the affected cells break down to form vesicles between the horny layer (as roof) and corium (as floor). Small vesicles coalesce with their neighboring ones forming larger vesicles which become filled with tissue debris and white cells. Similar lesions also occur at the same time in the mouth and esophagus. Due to the absence of a horny layer in the mucous membrane, the mucosal vesicles rupture early in the disease and shed virus into the secretion before the skin lesions become infectious. The infected epithelial cells of the skin and mucous membrane lesions show the typical intracytoplasmic eosinophilic Guarnieri inclusions.[14]

Smallpox is generally transmitted by direct contact; however, indirect transmission such as to laundry workers through infected bed clothes has also been observed. The disease, however, is less contagious than influenza or measles. The virus can survive in the dry state for months as evidenced by outbreaks of this disease in England that were traced to infected cotton imported from Egypt.

The disease has not occurred in the United States since 1949 when eight cases were diagnosed in the lower Rio Grand Valley in Texas. Prior to that, a traveller from Mexico caused 12 cases (2 fatal) in New York City in 1947. In Europe, however, many importations of smallpox have occurred since 1950. More than half of these cases were acquired by contact transmission in hospitals. An example of such importations occurred during January 1970 in Meschede, Federal Republic of Germany, in which the index case had returned from West Pakistan. Nineteen additional cases through two generations of spread (17 in the first and 2 in the second) were observed; four of these cases were fatal. Moreover, laboratory accidents have also caused outbreaks; one such accident at the London School of Hygiene and Tropical Medicine in 1973 caused four cases with two deaths. The index case, a female laboratory worker, did not work with smallpox virus but was exposed to it.

As the risk of acquiring smallpox in the United States became essentially zero in late 1960s, routine vaccination of children here was discontinued in 1971. In 1976, routine smallpox vaccination of United States hospital employees was likewise discontinued. The above policy was recommended by United States health officials because, first, vaccination was no longer necessary, and second, there is a finite number of complications associated with this immunization. It has been estimated that following primary vaccination, the risk of death is about one per million; that of hospitalization with encephalitis, eczema vaccinatum, or progressive vaccinia is about ten per million; and that of a serious complication including eczema vaccinatum, accidental implantation of vaccinia on the eye, or superinfection of a variety of skin conditions approaches 1000 per million.[15] However, United States Center for Disease Control (CDC) reported in June 1979, that during 1978 more than 4.4 million doses of smallpox vaccine were distributed in the United States. The vaccine has been needlessly administered to international travellers and, without any demonstrable benefit, used for the treatment of herpetic infections. Many serious and even fatal reactions to such smallpox vaccinations have been reported by CDC; the last fatal one — reported on March 14, 1980 — involved a 7-month-old child who was vaccinated because of a three-month history of recurrent mouth ulcers suspected of being caused by herpes simplex virus.

The WHO Global Eradication of Smallpox

The concept of eradicating this disease was conceived by Jenner himself who wrote in 1801, "it now becomes too manifest to admit of controversy that the annihilation of the smallpox, the most dreadful scourge of the human species, must be the result of this practice (vaccination)." Consequently, data collected in recent years from extensive worldwide investigations on smallpox indicated that the global eradication of this disease was achievable. This was based on the findings that, first, smallpox is a specifically human disease with no known animal reservoir; second, the disease occurs only as an acute infection in which the infected person either dies or recovers with lifelong immunity and without recrudescence; and third, there exists a very effective vaccine for protection against smallpox. However, Jenner's original hope for eradicating smallpox remained frustrated until 1950 when Dr. F. L.Soper, Director of the Pan American Sanitary Bureau, pro-

posed, at the Third World Health Assembly of the WHO, a program for smallpox eradication in the Americas. In the following years, however, beyond passing pious resolutions in annual meetings, little progress was made. In 1958 at the 11th World Health Assembly, the Soviet Union boldly proposed the adoption of the principle of global smallpox eradication as a policy. This was adopted by the assembly; however, preoccupation of many member countries with malaria eradication, unavailability of sufficient suitable vaccine doses, and inefficient budget hindered the progress of the program. In 1965 at the 18th World Health Assembly, the member countries took a much stronger position and thus, a more realistic budget was recommended. In the period between 1965 and 1968, three essential elements of the eradication program — availability of a stable vaccine of good quality, a more efficient vaccination method, and a more effective strategic approach — were worked out by the WHO experts.

In regard to the vaccine, 200-300 million doses were needed. As inhabitants of remote areas of tropical regions were also to be vaccinated, the use of a stable freeze-dried vaccine became essential. Donations of such vaccine were made by the Soviet Union (140 million doses annually), the United States (40 million doses annually), and some 20 other countries. Two selected international reference centers (in Canada and The Netherlands) were charged with testing the donated vaccines for potency, stability, and purity in accordance with the WHO standards. As to the method of vaccine administration, a device developed in 1968 by Wyeth Laboratories in the United States, which consists of a bifurcated needle for the multiple pressure technique, was universally adopted. The third element — strategy — had also to be worked out. The concept of mass immunization, originally proposed to include 80 per cent of the population, proved ineffective as smallpox persisted in such immunized populations. Since vaccination of 100 per cent of the population was virtually impossible to achieve, a shift in strategy which emphasized detection and containment of outbreaks was adopted. The above three modifications proved, as evidenced by the results, quite effective.

In January 1967, when the intensified global smallpox eradication program was started, the disease was present in at least 42 countries of which 33 were considered endemic areas (the latter in Asia, Africa, and South America involving a population of more than 1,200,000,000). Officially 131,160 cases of smallpox were reported in that year but it is believed that the actual number of cases was at least 2,500,000. The aim of the program was to eradicate smallpox from the entire world in 10 years. This goal has been achieved as the incidence of this disease declined rapidly and the last naturally occurring smallpox was in a Somalian in April 1977. The WHO, however, required two years of effective worldwide surveillance with negative results following the last recognized case before certifying the global eradication of smallpox. Thus, on October 26, 1979, the WHO declared that smallpox has been eradicated from the entire world. Later, victory over smallpox was officially marked by solemn ceremonies at the Palais des Nations in Geneva on May 8, 1980.* The credit for this phenomenal achievement deservedly belongs to the thoughtful and dedicated field workers, both national and international, who worked out remarkable solutions to very difficult problems with due consideration of the local circumstances.[11, 16-18]

The Somalian case is believed to be the last naturally occurring case of smallpox. The disease, diagnosed as variola minor, occurred in a 23-year-old male hospital cook from Merka, Somalia, named Ali Maow Maalin, who apparently was infected during a three-minute ride in a Jeep that was transporting two children with smallpox. The victim developed a rash on October 26, 1977, and recovered uneventfully without transmitting the disease to anyone else.

Many smallpox scares have been rumored since October 26, 1977. Special WHO teams have investigated all of these scares; all have proved false. The latest scare, which made worldwide headlines, concerned a 32-year-old Italian engineer, Umberto Moretti (most recently vaccinated in 1970), who developed smallpox-like symptoms on April 12, 1980, after returning from a trip to Japan, Indonesia, and Singapore. Although his illness was diagnosed clinically as chickenpox, electron microscopic study at an Italian laboratory reported poxvirus-like parti-

* In December 1979, the Global Commission for Certification of Smallpox Eradication declared that the world is now in the post-smallpox era, that no one, except investigators at special risk, should be vaccinated and that requirements for smallpox vaccination certificates at national frontiers throughout the world should be abolished. However, as a form of insurance against the likelihood (which is now considered to be negligible) of reintroduction of smallpox from laboratories or from natural or animal reservoirs, the Commission recommended that vaccine stocks to vaccinate 200 million people be maintained by the WHO indefinitely. Dr. Halfdan Mahler, Director General of the WHO has suggested that October 26 be officially designated as "Smallpox Zero Day." Moreover, a special WHO multilanguage poster, distributed worldwide, announces a $1000 reward for the first person who reports a confirmed case of smallpox acquired by person-to-person spread.

cles in the patient's skin lesions. However, further examination of the skin-lesion material by electron microscopy and chick embryo inoculation as well as complement fixation tests in Paris and at CDC indicated the involvement of the chickenpox virus.[19]

Poxvirus Disease After WHO Global Eradication

Following the global eradication of smallpox, two potential sources of poxvirus infection of humans have remained. The first is accidental infection with laboratory smallpox virus stocks, and the second, infection with animal poxviruses. The recent laboratory-associated infection in 1978 at Birmingham University Medical School in Britain is a vivid example of the first type. Mrs. Janet Parker, a 40-year-old medical photographer in the Department of Anatomy, worked in a darkroom located on the floor above a research laboratory where a comparative study of smallpox and whitepox viruses was performed by 40-year-old virologist Dr. Henry S. Bedson (the son of the late Sir Samuel P. Bedson) of the Department of Medical Microbiology. On July 25, 1978, Mrs. Parker spent most of the day on the phone in an office next to her darkroom ordering photographic supplies before the end of the financial year on July 31, 1978. While telephoning, she would have been close to an ill-fitting inspection panel of the service duct linking this office to Dr. Bedson's animal poxvirus laboratory. The inspection panel of the service duct in this laboratory was also ill-fitting. Off the animal poxvirus laboratory was a small laboratory where intensified research on, among other poxviruses, the Abid strain of variola major virus (isolated in 1970 from a 3-year-old boy in Pakistan) was proceeding. Very large quantities of virus were being handled in this laboratory. Bedson's laboratory was due to close at the end of 1978 as it had not been approved by the WHO as one of the few laboratories to continue work with smallpox virus after global eradication. In May 1978, a three-man WHO team inspected Bedson's laboratory and found that the physical facilities and laboratory procedures were far from satisfactory and clearly below the WHO standards and recommended either upgrading or closure at the earliest possible date. However, Bedson was regarded as a conscientious and experienced virologist of considerable worldwide repute and thus no peer pressure was brought on him to abide by the WHO team's recommendation. In the meantime, intensified work with the smallpox virus was being pursued by Dr. Bedson and his two assistants in an attempt to complete their research by the end of 1978.

Mrs. Parker, last vaccinated in 1966, became ill on August 11, was admitted to the hospital on August 24, her illness was diagnosed by electron microscopy as smallpox on August 25 (ironically by Dr. Bedson himself), and the Abid strain of smallpox virus was isolated from her on August 27, 1978. On September 11, 1978, Mrs. Parker died of smallpox in a Birmingham isolation hospital. As the other part of this double tragedy, Dr. Bedson had already died of self-inflicted throat wounds on September 6, 1978. A suicide note said, "I am sorry to have misplaced the trust which so many of my friends and colleagues have placed in me and my work." Mrs. Parker's mother and close contact, Mrs. Helen Witcome, was prophylactically vaccinated on August 25 and administered vaccinia immune globulin and methisazone. However, she developed a mild modified smallpox and recovered uneventfully. Thirty-nine close and 196 casual contacts of Mrs. Parker and her mother were placed under surveillance. One of these, a 20-year-old British woman, traveled to a North Dakota farm on August 18, 1978. Daily surveillance revealed no infection in any of these contacts.

The British Department of Health and Social Security appointed a team, headed by Professor Reginald A. Shooter, to investigate the laboratory-associated smallpox at Birmingham. This investigation resulted in the Shooter report which, considering the physical and procedural circumstances described above, indicated that Mrs. Parker was probably infected with the Abid strain of smallpox virus while telephoning on August 25, 1978. The virus apparently escaped from the small research laboratory into the animal poxvirus laboratory and from there to the service duct through the ill-fitting inspection panel and finally reached Mrs. Parker as she was talking on the phone (the virus here again coming out of the common service duct through the ill-fitting inspection panel). Ironically, the 1966 outbreak of smallpox, which affected 73 people with one death in Birmingham, started from the same source with the first victim being a photographer who had exactly the same position as Mrs. Parker. Previously, a hospital outbreak in Meschede, Federal Republic of Germany, was associated with the ability of smallpox virus to transmit from one floor to another via external air currents (see above).

The Health and Safety Executive issued a summons against the University of Birmingham for failing to protect the health of its employees. However, the Birmingham magistrates dismissed the charges largely on the basis that a number of prominent

smallpox experts (*e.g.* Professors Alan Downie, K. R. Dumbell, and K. McCarthy) challenged the thesis presented by the Shooter report and discounted the likelihood of airborne spread of the virus from the smallpox laboratory. The University of Birmingham has also issued its account challenging the Shooter report. At this writing, the case has not yet been definitely settled.[20-23]

Currently global efforts are being made to restrict all remaining smallpox virus stocks to only four WHO Collaborating Centers, *i.e.* CDC in Atlanta, Georgia, United States; the Research Institute of Viral Preparations, Moscow, USSR; the Rijksinstitut, Bilthoven, The Netherlands; and the National Institute of Health, Tokyo, Japan. However, many laboratories both in the United States and elsewhere, have so far refused to relinquish their smallpox virus stocks.

The second source of human infections — animal poxviruses — has become of considerable concern in recent years.* As smallpox virus is highly species-specific, it can only infect certain primates; however, infection in these hosts cannot be serially passed by contact transmission. Thus, it is believed that there is no simian reservoir for the smallpox virus. However, two other poxviruses of mammals — monkeypox and whitepox viruses — are capable or potentially capable of infecting humans. The monkeypox virus was first isolated in 1958 in Copenhagen from cynomolgus monkeys with a generalized pox disease. Later several outbreaks of monkeypox occurred, both in Europe and the United States, among monkeys imported from Asia and Africa. Human infection with the monkeypox virus, which is clinically indistinguishable from smallpox, was first recognized in 1970. Since then at least 36 sporadic cases have occurred in West and Central Africa; the last case occurred in a 35-year-old man in Nigeria who developed a rash on November 24, 1978. Only in two cases was the possibility of human-to-human transmission reported. Thus no epidemiological significance is given to this virus. In addition to African non-human primates, certain rodents of the region may serve as reservoirs. The virus is readily differentiated from the smallpox virus.[16, 18]

In 1964, two poxviruses were isolated from monkey kidney tissue cultures prepared in The Netherlands from kidneys of monkeys imported from Asia that had been in contact with African monkeys during transportation. The viruses were distinguishable from animal poxviruses but resembled the smallpox virus very closely. Subsequently, four more viruses with the same properties were isolated from a chimpanzee, a sala monkey, and two African rodents (Zaire). In contrast to monkey poxvirus, these viruses produce white pocks on the chorioallantoic membrane of the developing chick embryo, and have therefore been named whitepox viruses. Experimentally, they cause a generalized disease with rash in Cercopithecus monkeys. As these viruses are indistinguishable from smallpox virus by currently available laboratory procedures, they remain both a threat and a puzzle. However, to this writing, no human infection has been reported in areas where these viruses have existed.[16, 18, 24]

* The family of Poxviridae which contains the largest viruses (230 x 300 nm) with a DNA core and a complex structure includes two subfamilies, namely, Chordopoxvirinae and Entomopoxvirinae. The first includes genus Orthopoxvirus in which all poxviruses mentioned in this review are included. Biological and serological methods have indicated extensive antigenic interrelationships among orthopoxviruses; a recent study has classified them into the following three intra- and inter-related groups: (A) vaccinia, smallpox, alastrim, whitepox, and Lenny pox; (B) buffalopox, camelpox, mousepox (ectromelia), and MK-10 pox; (C) cowpox, monkeypox, and elephantpox. More work is needed for a universally accepted classification of these viruses. (See *Arch. Virol.* 54:143-145, 1977.)

Acknowledgement

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Case Reports

NON-HODGKIN'S LYMPHOMA OF THE PROSTATE

Although non-Hodgkin's lymphoma may involve any organ of the body, prostatic involvement, particularly at presentation, is a distinctly unusual event. Several reviews have documented the uncommon involvement of the prostate by non-Hodgkins's lymphoma. Richmond and associates, in an autopsy series of more than 400 patients, found the prostate to be involved in 11-23 per cent of cases with reticulum cell sarcoma or lymphosarcoma.[1] In a review of approximately 200 cases of lymphosarcoma, Sugarbaker and associates noted that secondary prostatic involvement was rare.[2] Lastly, Molander and Pack found evidence of prostatic involvement in only four of 378 male patients with non-Hodgkin's lymphoma.[3] In this report, we discuss a patient with non-Hodgkin's lymphoma (lymphocytic lymphoma) who presented with severe obstructive uropathy.

Case Report

A 57-year-old male presented to Truman Medical Center with a history of several months of progressive abdominal distention, nocturia, inability to void, and multiple urinary tract infections. During the previous three months the patient had experienced a weight loss of 25 pounds. There was no past history of kidney disease. Physical examination was unremarkable except for bladder distention to the umbilicus and an enlarged prostate which was firm with no nodules. Laboratory tests revealed the following abnormalities: blood urea nitrogen (BUN), 120 mg/dl; serum creatinine, 7.0 mg/100 ml; and a high anion gap metabolic acidosis. Creatinine clearance was 22 ml/min. Chest x-ray and acid phosphatase were normal. An intravenous pyelogram showed bilateral hydronephrosis with possible polycystic kidneys. CAT scan of the abdomen confirmed the bilateral hydronephrosis with no evidence of retroperitoneal masses or other abnormalities. A transurethral resection of the prostate was performed. Histologic examination revealed lymphocytic lymphoma. Following surgery the patient experienced a mild diuresis with renal function tests returning to BUN, 61 mg/dl and creatinine, 2.8 mg/100 ml. Urinary symptoms were much improved. Several months after discharge the patient returned complaining of general fatigue, weakness, and weight loss. Diffuse lymphadenopathy and splenomegaly were present. Repeat laboratory test results indicated widespread dissemination of the lymphoma; tissue confirmation was obtained from biopsies of femoral nodes and bilateral iliac crest bone marrow biopsies. BUN was 44 mg/dl and serum creatinine, 7.6 mg/100 ml. Repeat CAT scan revealed bilateral hydronephrosis and retroperitoneal masses thought to represent lymphomatous involvement of retroperitoneal nodes. Chemotherapy was initiated, and after several weeks the patient was clinically improved with BUN, 33 mg/dl and serum creatinine, 3.2 mg/10 ml.

Discussion

The clinical presentation of primary non-Hodgkin's lymphoma of the prostate is difficult to distinguish from other prostatic diseases producing urinary obstruction, particularly benign prostatic hypertrophy. The latter is an illness afflicting individuals mostly after the age of 40 years; thus urinary obstruction in the younger patient should arouse suspicion of other causes of obstruction. Physical examination and laboratory results may be normal if the lymphoma is localized within the prostate. In many instances, diagnosis may be made only during prostatic surgery with the preoperative diagnosis being benign prostatic hypertrophy. If a non-Hodgkin's lymphoma is found, proper staging should be done postoperatively. If no evidence of generalized involvement is found, surgical treatment may be curative, and careful follow-up is advised. If generalized involvement is found, appropriate radiotherapy or chemotherapy is indicated. Although rare, primary involvement of the prostate with non-Hodgkin's lymphoma indicates that lymphoid tissue is present in the prostate and that lymphomatous involvement of the prostate should be included in the differential diagnosis of lower urinary tract obstruction.[4]

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BILATERAL EXTERNAL OPHTHALMOPLEGIA

Since the first description by Jewett *et al.*,[1] several reports have appeared in the literature indicating usefulness of brainstem evoked responses (BER) in diagnosis of subclinical brainstem lesions. The importance of this procedure in diagnosing coma secondary to drug ingestion, multiple sclerosis, acoustic neuroma, and hearing defects has been well established.[2-5] Progressive external ophthalmoplegia from nuclear lesion secondary to vascular insufficiency in the territory of supply of the basilar artery is a rare lesion without any other concomitant brainstem structure involvement. We describe a patient with ophthalmoplegia from a nuclear lesion in which BER obtained via auditory click stimuli helped in establishing diagnosis.

Case Report

A 70-year-old white male presented with a history of good health until a year earlier when he was seen elsewhere with complaints of intermittent diplopia, dizziness, and occasional vertigo. Bilateral sixth nerve palsy was noted, and extensive neurological workup — including computerized axial tomography scan — was reported to be normal. Tensilon and thyroid function tests performed at that time were reported to be normal. Subsequently he was discharged, but a year later was admitted to Neurology Service with similar complaints. Results of the patient's general physical examination were unremarkable, except that he was noted to be mildly obese. Neurological examination revealed the patient to be alert, oriented, and without any speech impairment. Bilateral ptosis was noted, pupils were 3 mm, equal, and reactive to light. Fundi showed slight temporal pallor, and the patient was unable to move his eyes in any direction. Visual acuity was OS 20/30 OD 20/40, and visual fields were normal. The remainder of the neurological examination was within normal limits. Laboratory studies, electrolytes, fasting blood sugar, and serum chemistries were normal. Elec-

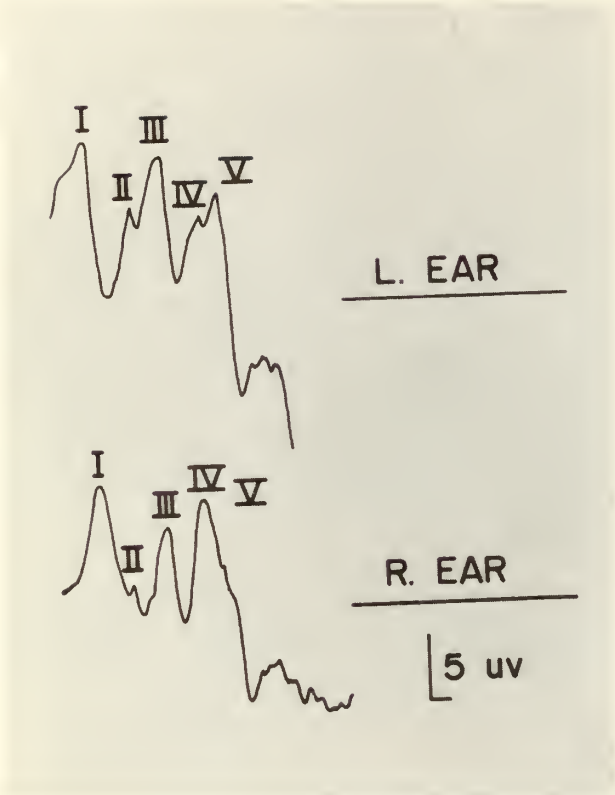


Figure 1. Brainstem evoked responses (BER) from patient, showing increased interpeak latencies of I-III and IV-V responses bilaterally.

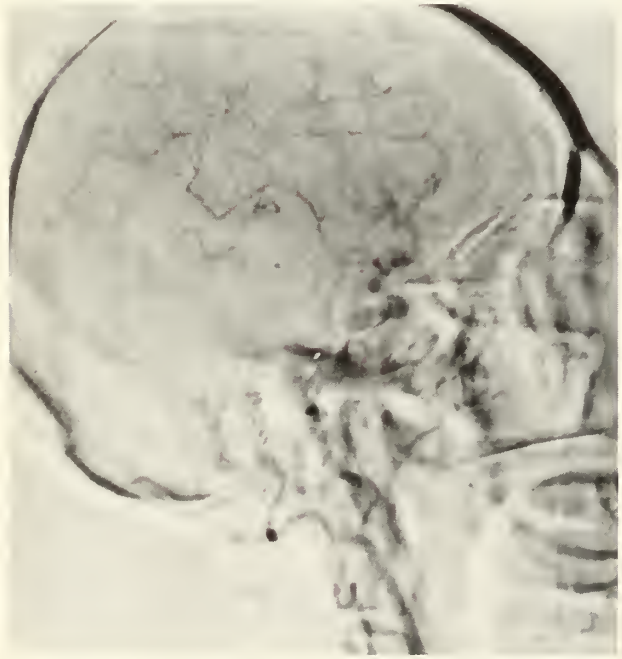


Figure 2. Bibrachial angiography showing occlusion of rostal third of basilar artery (arrow).

troencephalogram, radionuclide brain scan, and computerized axial tomography scan were normal. Tensilon and thyroid function tests were also within normal limits. BERs were obtained from auditory click stimuli using TECA 4 fiberoptic electromyography module, DAV 6 averaging computer, CK 6 click stimulator, and AA6 MK III amplifier at 65 Db SL. The click rate was 20/sec, and 2000 clicks were averaged monaurally from both ears; the entire procedure lasted four to six minutes. The results indicated that the patient had prolonged latencies bilaterally I-III and III-V interpeak latencies *(Figure 1)*. Cerebral angiography revealed occlusion of the rostral third of the basilar artery *(Figure 2)*.

Discussion

Jewett *et al.*, in 1971 described auditory evoked far fields averaged from the scalp of humans and found to consist of I-VIII waves. Wave V, with a latency of 5.1 msec and an amplitude of 0.6-1.4 IV was the most easily identified in all subjects. Wave I showed the same starting and peak latencies as N, recorded simultaneously from the ear canal. This, together with other evidence, suggests that Wave I is generated by the action potential of the eighth nerve. By analogy and from similar recording from animals, Wave II probably generates from cochlear nuclei, III from the superior olivary nuclei, IV-V at lateral leminiscus and inferior collicular level, VI and VII at the medial geniculate body and thalmic level respectively.

It has been found that these responses decrease in latencies from childhood (up to 2 years) to adulthood,[6] their latencies vary with the sound level, and are unaltered by drugs or sleep.[7] Interpeak latencies represent the time auditory stimuli travel in the brainstem. In this patient, I-III interpeak latency was 2.3 msec (normal 2 msec ± 0.1 msec), and III-V interpeak latency was 2.4 msec (normal 2.1 msec ± 0.1 msec). Amplitude of the responses was low *(Figure 1)*.

In this case, the patient developed bilateral external ophthalmoplegia from vascular infarction of the third nerve nuclei secondary to basilar artery occlusion. External ophthalmoplegia from nuclear infarction is uncommon and has been rarely documented in the past.[8, 9] Auditory pathways probably become affected in a similar way from diffuse arteriosclerosis.

In the above study, BER abnormalities clearly indicated the lesion to be in the brainstem; this diagnosis was subsequently proven by arteriography.

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RECTAL CARCINOMA ASSOCIATED WITH GLIOBLASTOMA MULTIFORME OF THE BRAIN

Carcinoma of the rectum and glioblastoma multiforme (astrocytoma, grade III or IV) are both extremely rare in younger age groups. When both appear in the same patient, a genetic predisposition is suspected.

Case Report

A 15-year-old male was hospitalized in June 1976 with a diagnosis of carcinoma of the rectum. For three weeks prior to admission he had experienced nausea, vomiting, and diarrhea. Evaluation revealed a mass in the rectum. Histories of both

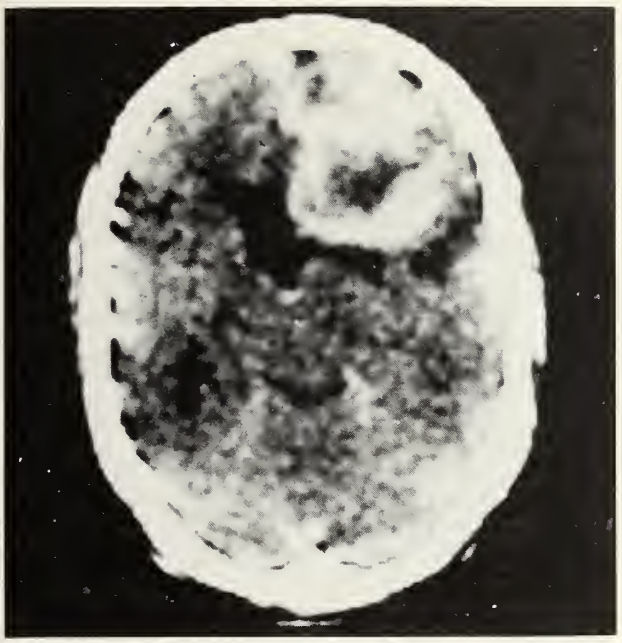


Figure 1

parents were negative for gastrointestinal and CNS disease or symptoms, but there was a strong history of gastrointestinal tract cancer in several paternal aunts. The patient's sister had died of carcinoma of the colon at the age of 19 years. (She had undergone surgery for removal of multiple polyps prior to her death.)

Examination revealed a weak, bedridden boy with sepsis manifested by high spiking temperatures and chills. He had numerous acneiform lesions over the face and, to a lesser extent, over the entire body. When sepsis was brought under control in July, he underwent abdominal-perineal resection. The surgical specimen (40 cm) did not show any polyps.

Three weeks following surgery the patient began experiencing continuous bitemporal headaches without associated nausea or vomiting. Work-up, including CT scan of the head, revealed a mass lesion in the right frontal lobe *(Figure 1)*. A right frontal craniotomy was performed but only a subtotal resection of the tumor was possible. The pathology report was astrocytoma, grade IV. Two weeks post craniotomy, radiation therapy to the brain was initiated, and the patient received 4976 rad in 22 treatments during a period of 81 days. During the course of radiotherapy, he developed severe fungal stomatitis which responded to conservative therapy. However, his condition gradually deteriorated, and he died in February 1977.

Discussion

This case is of interest because the association of these two tumors in a young patient is rare. All similar cases previously reported involved familial polyposis or manifestations such as thyroid cancer or Von Recklinghausen's disease.[1-7] In our case, the patient presented with generalized acneiform lesions over the entire body, a condition not typical of neurofibromatosis.

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HEMOSPERMIA AND HYPERTENSION TWO CASE REPORTS

Hemospermia — the presence of blood in the male ejaculate — is an uncommon problem in the practice of internal medicine. Literature reviews and case reports have been few and primarily in the urologic realm.[1, 2] These articles have emphasized the association of hemospermia with tuberculosis or prostatic disease. Little mention is made of the possible relationship of hypertension and hemospermia. In a study of hemospermia by Yu and associates, 65 cases of this condition were reviewed with no patients having simultaneous hypertension.[3] Only in the review by Ross is hypertension mentioned as a possible significant cause of hemospermia.[4] In this report, we present two cases of severe hypertension and hemospermia with correction of the hemospermia occurring after the reduction of the elevated blood pressure.

Case One: A 31-year-old white male was admitted to the hospital for control of hypertension. Past medical history and review of symptoms were negative except for the presence of blood in his ejaculate, described as bright red, for the previous eight months. There were no associated genitourinary tract symptoms. The patient was not receiving any medication. Blood pressure was 200/140 mmHg. Examination of the genitourinary tract was unremarkable. A tuberculin skin test was negative. Semen analysis revealed bright red blood. Sperm count, morphology, and motility were normal. Semen cytology was not remarkable. Culture of semen for bacteria and acid-fast organisms was negative. Urinalysis was within normal limits and a urine culture was negative. Prostatic massage revealed no bacteria. Prothrombin time, partial thromboplastin time, and platelet count were normal. Anti-hypertensive drug therapy, including furosemide, propranolol and prazosin, resulted in a decline in blood pressure to 160/90 mmHg. Continued control of blood pressure was associated with alleviation of hemospermia within one month. Follow-up of this patient during a period of several months has shown continued absence of blood in his ejaculate with no new genitourinary symptoms. There has been no change in his pattern of sexual activity.

Case Two: A black male, 45 years of age, was admitted to the hospital for control of hypertension. Past medical history and review of symptoms were negative except for the presence of blood in his ejaculate, described as dark red, for the past two months. There were no other symptoms referrable to the genitourinary tract. Medication upon admission included periodic furosemide, propranolol, and hydralazine. Blood pressure was 220/160 mmHg. The remainder of the physical examination was non-contributory. Examination of the genitourinary tract was unremarkable. Semen analysis revealed gross dark red blood. Sperm count, morphology, and motility were normal. A tuberculin skin test and a smear of semen and urine sediment for acid-fast bacilli were negative. Results of urinalysis and urine culture were negative. Prostatic massage was negative for bacteria. Prothrombin time, partial thromboplastin time, and platelet count were normal. Chest x-ray was negative for tuberculosis. Anti-hypertensive drug therapy — including furosemide, propranolol and prazosin — resulted in a decline in the patient's blood pressure to 150/100 mmHg. Within a month of continued blood pressure in this range, the ejaculate cleared of blood. Follow-up of this patient during a period of several months has revealed no further genitourinary abnormality. Blood has consistently been absent from his ejaculate. There has been no change in the pattern of sexual activity.

Discussion

Hemospermia may be the presenting symptom of a wide variety of pathologic entities. The differential diagnosis of this

condition includes prostatis, carcinoma of the prostate, carcinoma of the seminal vesicles, genitourinary tuberculosis, a systemic blood dyscrasia, and sexual excesses.[4] A search of the literature in internal medicine did not reveal any reports of hemospermia associated with pronounced hypertension. Most studies have been reported from departments of urology and have stressed the strong clinical association of hemospermia with tuberculosis when an underlying pathologic condition is found.[5] In fact, the majority of cases are idiopathic or primary.[6] There are several theories that might explain idiopathic hemospermia. Shropshire believed that sudden emptying of the distended seminal vesicle during ejaculation caused bleeding.[7] In another early review, Pelouse concluded that the pathologic lesion causing hemospermia was rupture of engorged veins in the mucous membranes of the posterior urethra.[8] Several other authors have postulated that damaged blood vessels in the seminal vesicles are the underlying lesion in hemospermia.

The clinical courses of the two patients presented in this report indicate that pronounced hypertension may cause hemospermia. It is possible that the hemospermia was due to a necrotizing ateriolitis of the seminal vesicles or other anatomic areas involved in the production and transport of semen. There was strong laboratory evidence against an infectious cause. In addition, there was no clinical or pathologic evidence of prostatic disease. The clinical presentation and laboratory values did not indicate a bleeding disorder. There was no change in the sexual activity pattern of either patient. More importantly, the hemospermia disappeared and has remained absent with reduction of blood pressure. Generally, as with these two patients, there are no other signs or symptoms of genitourinary disorders associated with hemospermia. In the review of Tolley and Castro, all 18 patients with hemospermia as their sole genitourinary symptom had extensive urologic examinations that revealed no abnormality.[2] It was their conclusion that such patients do not require detailed investigation, whereas those with other urologic symptoms do. Similar conclusions have been reached by other investigators.[1] Our two case reports support the above studies. In addition, we recommend that before extensive urologic studies are undertaken in patients with hemospermia and hypertension, that the hypertension be corrected. If there are other signs or symptoms of genitourinary tract disorders in addition to hemospermia, or if reduction of the blood pressure does not alleviate the hemospermia within a month, further urologic investigation is warranted.

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RECURRENT MASSIVE GASTROINTESTINAL BLEEDING DUE TO GASTRIC LEIOMYOMA

Leiomyomas are benign interstitial tumors, growing submucosally or subserously on any part of the body where smooth muscle fibers exist. The most common locations are the gastrointestinal (GI) tract, retroperitoneal space, uterus, and bladder. Leiomyoma of the uterus constitutes one of the most common neoplasms in humans. Leiomyomas of the GI tract are the most common benign tumors, and occur in the following frequencies: stomach, 65 per cent; small intestine, 20 per cent; colon, 10 per cent; and esophagus, 5 per cent.[1]

Incidences

In 1961, Grigg[2] estimated that about 2,000-2,500 cases of simple and malignant GI leiomyoma had been reported. In 1944, Meissner[2] found gastric leiomyoma in 46 per cent of consecutive autopsies. Skandalakis,[3] in 1960, studied 1,000 cases of gastric tumors of muscular origin, 600 of which were benign. Gastric leiomyoma arises from the muscularis mucosa or the muscular layer of the stomach and constitutes from 40-50 per cent of benign gastric tumors.[3] Autopsy of patients from 40-50 years of age has shown gastric leiomyoma in 15 per cent.[4] Akerman[5] reported a 23 per cent incidence in a prospective autopsy study, and of all excised gastric tumors, 5 per cent are non-malignant.[6] Reported here is a case of recurrent massive gastric bleeding due to a large penetrating gastric leiomyoma.

Case Report

An 83-year-old white female presented at the emergency room in October 1978 complaining of fainting attacks, weakness, shortness of breath, vomiting, and passing blood. The patient was lethargic with blood pressure 100/60, despite a history of hypertension, and pulse was irregular.

The patient's past history included arthritis and coronary artery disease of long duration. She had been hospitalized two years earlier for massive upper GI bleeding which was attributed to acute gastroenteritis. The diagnosis of leiomyoma was missed by x-ray and gastroscopy, although a filling defect was mentioned by the radiologist. The patient received 28 units of blood during that hospitalization.

Laboratory and x-ray examinations on admission in 1978 included hemoglobin, 5.7; hematocrit, 20%; and WBC, 8,900 with normal differential. SMA 20 was normal except for low iron, 25 mg; sodium, 126; protein, 5.5; and albumin, 3.3. Chest and abdominal x-rays revealed a soft tissue mass in the right abdomen. Liver/spleen scan was normal. Upper GI x-ray showed a sliding hiatal hernia with mild reflux esophagitis, and edematous changes of the mucosal folds of the gastric fundus with possible superficial ulceration. (This x-ray was taken after informing the radiologist of the gastroscopic findings.) Barium enema testing showed abnormal findings at the upper sigmoid, with a suggestion of carcinoma or ulcerative colitis. Electrocardiogram revealed premature atrial contractions.

The patient received six units of blood. Gastroscopy revealed mucosal congestion with two ulcers and a submucosal bulge. Proctosigmoidoscopy revealed a benign polyp which was confirmed by biopsy. The patient was taken to surgery on November 1, where colon resection for carcinoma and bilateral salpingo-oophorectomy with removal of a large ovarian tumor and segmental resection of the stomach were performed. Postoperative complications included stress gastritis which responded well to conservative treatment, and arrhythmias which responded well to Pronestyl. She received total blood transfusions of 13 units. On November 13, hematocrit was 38% and hemoglobin, 13 gm/100 ml. The patient was dismissed on November 17.

Discussion

The clinical behavior of gastric leiomyomas depends on location, size, and whether the tumor extends inward or outward in the stomach. Their most distinctive clinical feature is the tendency to ulcerate and cause GI bleeding. Fifty per cent of gastric

leiomyomas present with ulceration of gastric mucosa, and 42 per cent of patients manifest massive GI bleeding.[7] Other patients will present with occult bleeding. Aspirin has been incriminated in initiating massive GI bleeding in a patient with gastric leiomyoma.[2]

The 1970 Connecticut study of the American board of Surgeons[5] reported 296 cases of massive GI bleeding; eight patients had gastric neoplasms, one of which was a gastric leiomyoma and another leiomyoblastoma.

Ochnsa and Janetos in their series (1965) reported a 42 per cent incidence of GI bleeding; Beard and colleagues (1968), 48 per cent; Skandalakis (1962), 55 per cent; and Thompson and Oyster (1950), 71 per cent.[2] The causes of upper GI hemorrhage are hemorrhagic gastritis, 47 per cent; chronic peptic ulcer disease, 22 per cent; benign and malignant tumors, 12 per cent; and leiomyoma, 1.2 per cent.[8]

The growing of the tumor is followed by necrosis toward its center, resulting in the creation of multiple cavities full of blood. The walls of the cavities often burst and hemorrhage.[9] The other mechanism of hemorrhage is erosion of the gastric mucosa by tumor growth in one or more sites causing bleeding that is usually massive, thus resulting in an ulcer that is usually chronic, intractable, and difficult to heal. Headache, fainting spells, vomiting, and collapse may portend imminent hemorrhage.

Symptoms of gastric leiomyoma are weight loss, epigastric pain, nausea, abdominal mass, vomiting, and anorexia.[5] Other complications of gastric leiomyoma include: necrosis, which can cause abscess, perforation, peritonitis, or hemorrhage; cystic degeneration of the tumor; hyalinidosis; and calcification. Malignant degeneration of gastric leiomyoma is rare, although some reports note that 20 per cent of all GI leiomyoma undergo malignant degeneration, 40 per cent cause hemorrhage, and 20 per cent cause obstruction.[9] The affliction can occur at any age; the youngest patient reported with gastric leiomyoma causing bleeding was 14 years of age.[10]

Treatment

Gastric leiomyomas should be surgically treated as soon as diagnosed. The surgical procedure may vary according to size, location, and clinical presentation. The procedure may vary from wedge resection to subtotal gastrectomy or subserosal shelling out of the tumor, with or without omentectomy. Incidental gastric leiomyomas should be removed to prevent complications.

Summary

Gastric leiomyomas are an unusual cause of upper GI bleeding (1-2%). They have no pathognomic signs or symptoms. The diagnosis depends on radiologic demonstrations of intraluminal or extraluminal mass, and in some cases gastroscopy may be the only way to obtain an accurate diagnosis. Leiomyomas cause bleeding by erosion of the gastric mucosa, thus causing ulcers that usually are superficial and may be multiple.

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LIFE THREATENING LACTIC ACIDOSIS IN STATUS EPILEPTICUS

Scanning the literature reveals only a few reports relating to lactic acidosis in seizure disorders. Orringer *et al.*,[1] has recently reported short-lived lactic acidosis complicating a single episode of grand mal seizure. However, acid base disturbances in status epilepticus have not been generally reported.

The two cases of status epilepticus presented here show that severe lactic acidosis — at times life-threatening — could complicate such a situation. This acute acid base derangement needs close monitoring and may require cautious and partial correction during the critical phase of metabolic acidemia.

Status epilepticus is usually defined as recurrent seizures without interim recovery of consciousness. Grand mal status may occur in about 3 per cent of epileptics and may be the first event in some patients. It may be precipitated in a known epileptic by events such as sudden termination of anticonvulsant therapy, intercurrent illness, other drugs, or alcohol abuse. Often, however, it occurs without apparent cause.[2]

Case One

A 56-year-old white male was admitted as an emergency case to the Intensive Care Unit in status epilepticus. The diagnosis was grand mal seizure, idiopathic origin, dating from 1965, and controlled with Dilantin, 400 mg/day. He had been seizure free since 1975. The patient was otherwise healthy, and previous medical evaluations were unremarkable. His wife, who found him unconscious, witnessed a series of rapidly recurring seizures three times in less than 15 minutes, and the same pattern continued during his transfer and in the emergency room — a total of seven in less than one hour. She also related a recent lack of compliance with his anticonvulsant medication, but denied alcohol or other drug abuse.

Physical examination on admission showed an unconscious, middle aged, normotensive white male who was apyrexial with regular heart rate of 108/minute and respirations of 26/minute. There were no signs of external injuries or meningeal irritation, and the fundi were normal. No localizing neurological signs were seen, and the rest of the clinical examination was unremarkable. Initial laboratory studies revealed sodium, 141 mEq/L; potassium, 3.9 mEq/L; chlorides, 101 mEq/L; CO_2, 4.7 mEq/L; anion gap, 37 mEq/L; blood sugar, 217 mgm/100 ml; and BUN, 11 mgm/100 ml. Arterial blood gases on two liters of oxygen were pH, 7.13; pO_2, 72.1; pCO_2, 34.9; HCO_3, 11.4; and base excess of −16.6. Serum was negative for ketone bodies (β-hydroxybutyric). Screening for drugs and alcohol was negative. Urine tests for sugar and acetone were negative. Other tests included serum lactate, 13.0 mEq/L (normal 0.5-2.2/L); serum pyruvate, 1.0 mgm/100 ml (normal 0-1.6 mgm/100 ml); and lactate/pyruvate ratio, 13. SMA-18 was normal including serum calcium and magnesium. Serum dilantin level was 3.3 μgm/ml (normal 10-20). Later investigations — including electrocardiogram, chest x-ray, skull x-ray, echoencephalogram, and brain scan — yielded normal results. Electroencephalogram was abnormal with right hemisphere slow wave disorder of maximum intensity in the right temporal region.

Emergency measures were instituted on admission to ensure free airways with adequate oxygenation. Seizures were controlled by intravenous diazepam. The patient received $NaHCO_3$, 44 mEq, at the critical phase of metabolic acidosis (pH, 7.13). Monitoring of arterial blood gases and blood chemistry showed gradual resolution of lactic acidosis without rebound alkalosis *(Table I)*. The patient gradually regained consciousness during a

TABLE I
SEQUENTIAL CHANGES IN ARTERIAL BLOOD IN CASE ONE

Time	*0*	*1h*	*2h*	*4h*	*24h*
pH	7.13	7.21	7.26	7.34	7.42
pO_2	72	60	63	65	76
pCO_2	34	40	42	40	31
HCO_3	11.4	15.9	17.2	21.4	22.9
Base Excess	−16.6	−10.7	−8.4	−3.1	−1.9

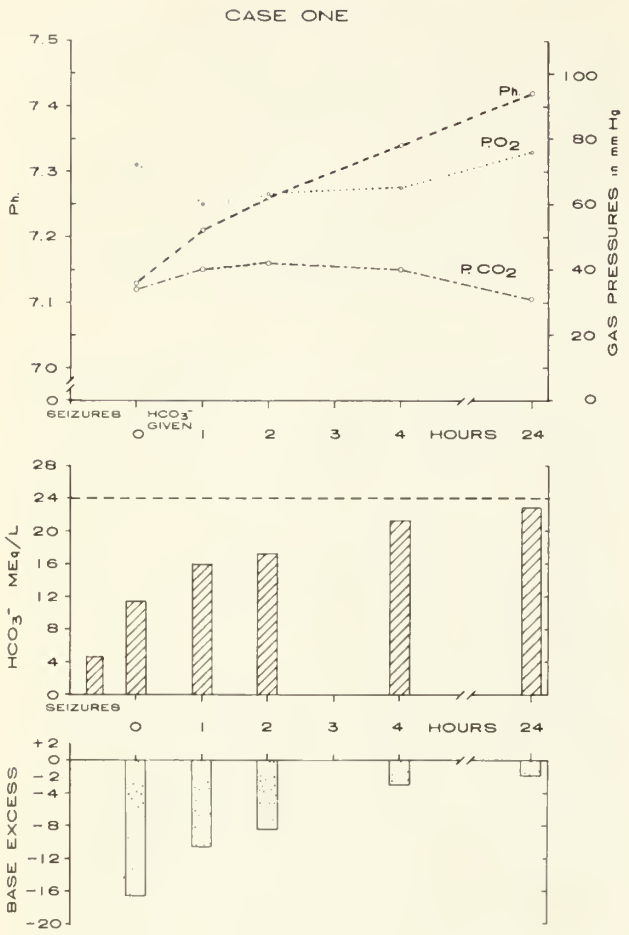


Figure 1A

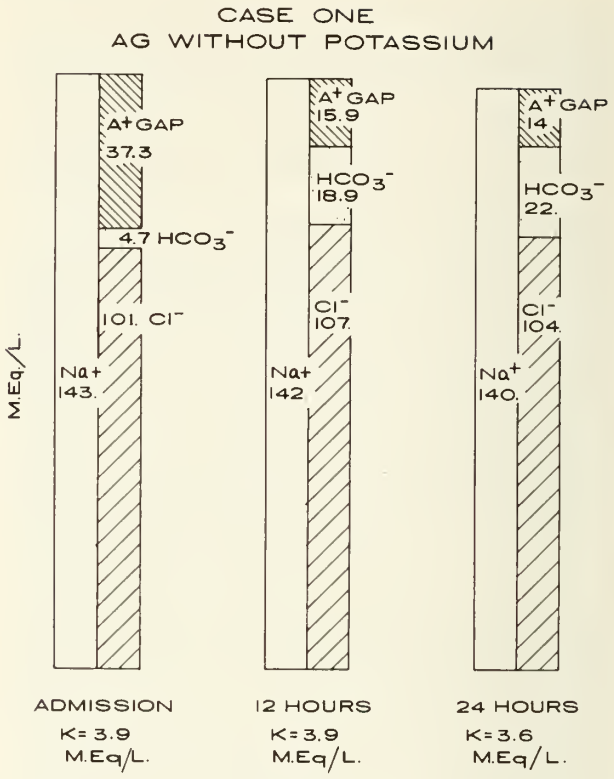


Figure 1B

period of three hours, and recovery was complete. The patient's treatment and progression are shown in Figure 1.

Case Two

A 28-year-old male nurse, employed by the Topeka Veterans Administration Medical Center, had undergone pre-employment physical examination and laboratory studies two months prior to admission. Findings were normal, and past medical history was non-significant. During a day shift, he suddenly lost consciousness with rapidly recurring grand mal seizures times four. Following emergency measures, the patient was transferred to the Intensive Care Unit. On examination he was found to be deeply unconscious without localizing neurological signs and the remainder of the clinical examination was unrevealing. However, sites of multiple needle punctures were detected over the extremities and buttocks.

Initial laboratory tests revealed sodium, 139; potassium, 4.1; chlorides, 101; and CO_2, 12. Anion gap was 26; blood sugar, 128; and BUN, 17. Arterial blood gases on three liters of oxygen were pH, 7.08; pO_2, 129; pCO_2, 43; HCO_3, 13.2; and base excess of −15.8. SMA-18 was normal. Serum testing for acetone and ketone bodies was negative. No salycilates and no alcohol were detected. Serum lactate level was 16.5 mEq/L (normal 0.5-2.2 mEq/L). Serum pyruvate level was 1.2 mEq/L (normal 0-1.6 mEq/L). Lactate/pyruvate ratio was 13.8. Drug screening was strongly positive for Demerol, and weakly positive for Talwin and barbiturates. Chest x-ray, electrocardiogram, complete blood count, electroencephalogram, and brain scan were all within normal limits. The impression was that of grand mal status with severe lactic acidemia complicating drug abuse and barbiturate withdrawal.

Grand mal status was managed with conventional therapy. The serious acid base derangement was considered life threatening, and correction of the base deficit was accomplished with sodium bicarbonate (44 mEq) administered when the pH was 7.08. During the critical phase, monitoring of arterial blood gases and blood chemistry showed continued resolution of lactic acidosis without rebound alkalosis *(Table II)*. However, a short-lived respiratory depression continued for a slightly longer period as a result of the narcotics. Recovery was complete without neurological deficit and the patient was discharged on the third day to a drug abuse program. The patient's treatment and progression are shown in *Figure 2*.

TABLE II
SEQUENTIAL CHANGES IN ARTERIAL BLOOD IN CASE TWO

Time	*0*	*15 min*	*1h*	*4h*	*24h*	*48h*
pH	7.08	7.12	7.29	7.35	7.38	7.37
pO_2	129	123	95.4	97	95	90.8
pCO_2	43.7	49	49	47	46	47.9
HCO_3	13.2	17.5	26	28	30	30.1
Base Excess	−15.8	−11.8	0	2.5	4.6	4.6

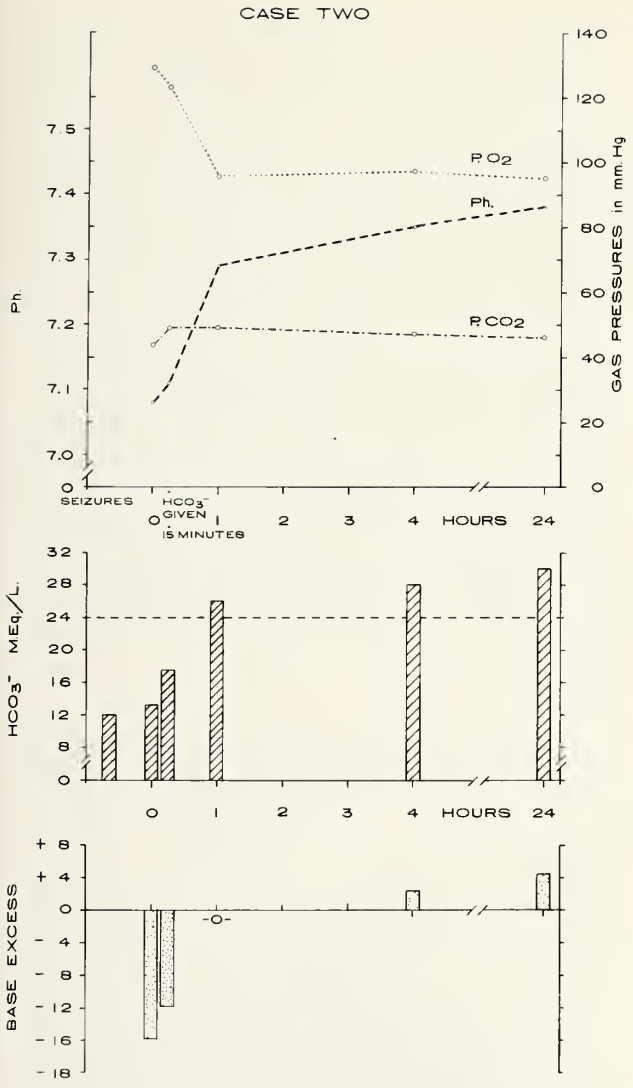


Figure 2A

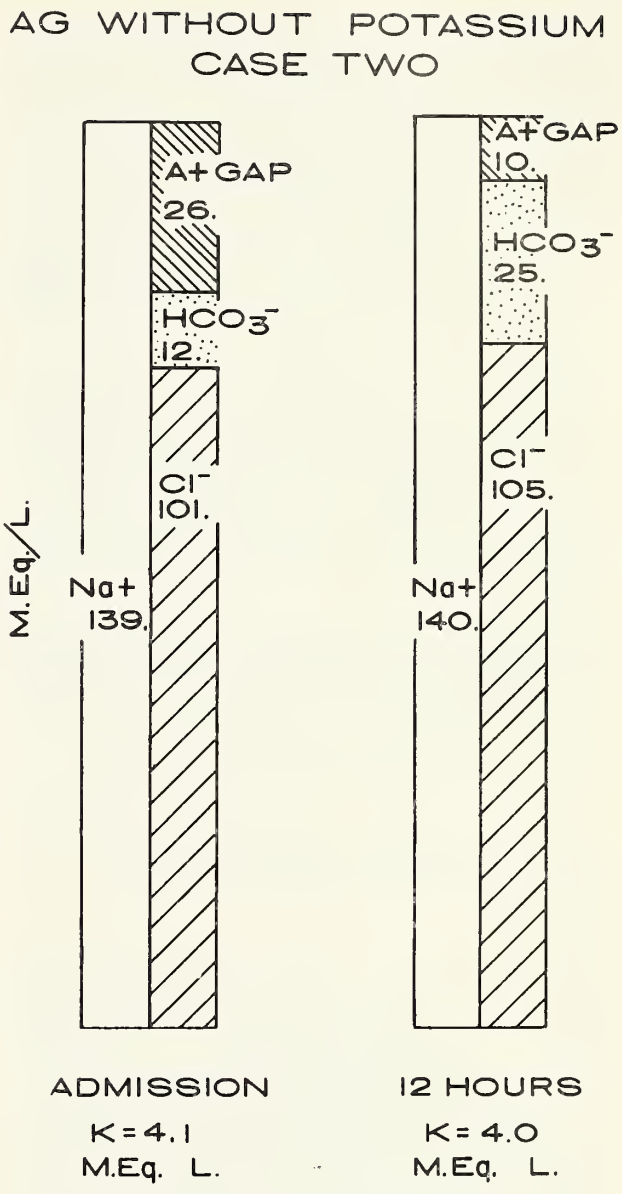


Figure 2B

Discussion

Major status epilepticus is a grave medical emergency in which the mortality rate can be as high as 21 per cent.[3] This has long been a major concern and a great challenge. Until 1970, the major clinical reviews in the literature[4, 5] have not mentioned convulsive disorders as a recognized cause of lactic acidosis. It was only in 1971 that Kulbermalten *et al.*[6] reported that spontaneous seizures in man are associated with acute metabolic acidosis secondary to lactic acid accumulation. In 1975, Brooks[7] demonstrated increased cerebrospinal fluid lactate levels following seizures. Orringer *et al.*[1] in 1977 reported short-lived lactic acidosis following one single grand mal seizure. However, no patients with recurrent seizures or status epilepticus were included in their study.

In the two cases of status epilepticus presented here, we found that profound degrees of lactic acidemia, reaching critical levels, complicates the situation. Our study has also shown that the time course for resolution is more prolonged than after a single grand mal seizure, and with cautious partial correction up to 7.20, recovery was complete without rebound alkalosis.

The pathophysiologic events after a single grand mal seizure are usually mild, transient, and tolerated by most patients. The lactate generated by the contracting muscles in the ictal phase is rapidly utilized when aerobic metabolism is re-established.[8] Hence, lactic acidosis is short-lived, and spontaneous resolution usually occurs within one hour after cessation of the seizure activity.[8, 9] In contrast, the metabolic stress in status epilepticus is more pronounced and persistent. The seizure activities are recurrent and sustained, and there is lack of seizure-free periods. This will favor anerobic metabolism. In such a situation there is more lactic acid formation with lack of utilization, which evolves into a state of progressive lactic acidemia. When this extends beyond the critical levels it will result in deleterious cardiovascular effects with hemodynamic instability. This compounds the picture by more tissue hypoxia and aggravation of the lactic acidosis. Thus, a life threatening vicious circle may evolve. Therefore, in a critical state where pH is below 7.20, a

life support measure of partial correction of profound acidemia and rapid control of seizures are essential. However, it should be emphasized that the patient's HCO_3 should not be restored to values greater than 12-14 mEq to prevent alkaline overshoot.

Despite the severe lactic acidosis in our patients, the serum potassium levels did not show any elevation. This was in agreement with the findings of Orringer[1] and others. This apparent discrepancy to other types of metabolic acidosis[10] may be explained by the fact that lactic acid penetrates cells rapidly leading to acidosis of both intracellular and extracellular compartments. This action results in a proportional decrease in pH on both sides of the cell membrane, requiring no shift of potassium from intracellular to extracellular fluid.

Conclusions

Profound acid base derangement in status epilepticus is due to rapid accumulation of lactic acid. Despite the severe lactic acidosis in our patients, the serum potassium level did not show any elevation. Monitoring of blood gases and chemistry are essential during the critical phase of acidemia; cautious and partial correction of profound metabolic acidosis (below pH 7.20) is needed to reduce the gravity of the life threatening situation.

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References may be obtained from the authors.

ACUTE DIGOXIN OVERDOSAGE: POTENTIAL ROLE OF COLESTIPOL THERAPY

Digitalis intoxication is a frequent iatrogenic complication associated with the use of cardiac glycosides in the treatment of heart disease. The frequency of intoxication ranges between 10-35 per cent in digitalized inpatients.[1, 2] In contrast to this frequency in long-term therapy, acute digitalis intoxication is infrequent. Acute intoxication is a medical emergency and a therapeutic dilemma. Management requires termination of the toxic exposure, administration of specific antidotes, and supportive therapy.[3] Recently interest has been shown in the treatment of digitalis intoxication by insoluble, nonabsorbable bile-acid-binding resins which interrupt the enterohepatic circulation of bile acids.[4] Since cardiac glycosides and bile acids have a similar chemical structure, bile-acid-binding resins should bind digitalis or digoxin in the intestine thus interrupting their enterohepatic circulation and resulting in a shortened elimination half-life. Contrary to early reports, the biliary excretion of digoxin has been found to be significant in healthy subjects suggesting that interruption of the enterohepatic cycle in digoxin intoxication might be beneficial.[5] In this case report we discuss an acute digoxin intoxication treated with colestipol, a relatively new steroid-binding resin, that causes a shortening of the digoxin elimination half-life.

Case Report

A 68-year-old white male with a history of hypertension and heart disease presented to the emergency room with a five-day history of nausea, vomiting, and diarrhea. Medication history included Lanoxin, 0.125 mg/day; Isordil, 10 mg four times/day; Aldactone, 25 mg four times/day; and Indocin, 24 mg three times/day. Approximately a week before admission the patient accidently began to take the Lanoxin tablets four times/day, mistaking them for the Aldactone pills. Blood pressure was 100/60 mm Hg; pulse, 40 and regular; temperature 36.1 C; and respiratory rate, 30/min. The patient was confused, disoriented, and in moderate distress. The remainder of the physical examination was noncontributory. Initial laboratory data included serum potassium, 6.8 mEq/L (normal 3.5-5.3 mEq/L); serum creatinine, 1.6 mg/dl (normal 0.7-1.4 mg/dl); serum magnesium, 2.6 mg/dl (normal 1.8-2.4 mg/dl); and digoxin level of 10.9 ng/ml (normal 0.8-2.1 ng/ml). An electrocardiogram revealed a nonparoxysmal junctional tachycardia with a rate of 40 that was resistant to atropine and that progressed to complete atrioventricular block. A diagnosis of acute digoxin intoxication was made. Treatment included insertion of a temporary cardiac pacemaker, kayexelate therapy, and supportive care. Colestipol, an insoluble copolymer of tetraethylenepentamine and epichlorohydrin, 10 gm initially and 5 gm/six hrs by mouth, was begun 12 hours after admission. The half-life of digoxin prior to colestipol therapy was 31.9 hrs; during colestipol therapy the half-life was reduced to 23.2 hrs, and after discontinuance of colestipol therapy the half-life rose to 33.5 hrs. Colestipol therapy was begun at a digoxin level of 8.7 ng/ml and was terminated at a digoxin level of 1.8 ng/ml. The patient recovered, and digoxin therapy was not reinstituted. Upon discharge the creatinine level was 0.9 mg/dl and the creatinine clearance was 76 ml/min.

Discussion

The treatment of acute digitalis intoxication may include measures designed to terminate exposure to the offending agent such as emesis, gastric lavage, and activated charcoal administration along with therapy designed to hasten elimination of absorbed drugs such as the use of digoxin-specific antibodies and charcoal hemoperfusion.[3, 6, 7] In addition, supportive care of electrolyte abnormalities or arrhythmias is crucial. However, several of these modalities are not readily available and are also potentially dangerous. A recent review by Warren and Fanestil disclosed that, despite a significant drop in the serum digoxin concentration, hemoperfusion removed less than 1 per cent of the total ingested digoxin in a case overdose.[8] In contrast, bile-acid-binding resins such as cholestyramine and colestipol are readily available and provide rapid action with minimal systemic side effects. In our patient the administration of colestipol appeared to reduce the elimination half-life of digoxin based on the elimination characteristics studied before, during, and after resin administration. A similar result was shown in a patient treated with colestipol in a review by Bazzano and Bazzano.[9] These same authors showed that, *in vitro,* the pressure of duodenal juice may decrease the glycoside-binding capacity of cholestyramine while not inhibiting the binding capacity of colestipol. From their results it appeared that colestipol may be the preferred binding resin in the treatment of acute digitalis intoxication. We believe that further studies into the clinical efficacy of colestipol in acute digitalis intoxication appear warranted.

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References may be obtained from the authors.

NON-OPERATIVE MANAGEMENT OF PSEUDOTUMOR CEREBRI CAUSED BY LATERAL SINUS THROMBOSIS

Widespread use of antibiotic therapy for the treatment of chronic otitis media and mastoiditis has made thrombosis of the lateral sinus a rare occurrence. Much of the literature on this subject is derived from the clinical experience in the pre-antibiotic era, and thus most authors indicate that surgery is mandatory in the treatment of this condition. We report here a case of lateral sinus thrombosis and secondary "otitic hydrocephalus" which was successfully treated without surgical intervention.

Case Report

An 11-year-old white female was in good health except for repeated episodes of otitis media which had occurred several times per year since the age of four years. In her most recent attack she was treated with an oral penicillin preparation for one week. Pain disappeared, but approximately one week after cessation of antibiotic therapy, ear pain and drainage recurred. At that time she had spiking fever to 40.6 C and consequently was given a repeat course of intramuscular penicillin for ten days. Approximately two weeks after onset of illness, she experienced pain behind the right eye, developed diplopia on lateral gaze, and began vomiting. On admission to Pediatric Neurology Service at the University of Kansas Medical Center, she was noted to be alert, oriented, and in no acute distress. Vital signs were stable and her temperature was 36.9 C. Neurologic examination revealed bilateral papilledema, with a left VI nerve palsy. The right tympanic membrane was thickened and gray-white with no landmarks visible.

Laboratory workup revealed the following: WBC, 11,000 with 90% segmented forms, 8 lymphs, and 2 mononuclear cells; sedimentation rate, 21; mycoplasma Pneumoniae titer, positive at 1 : 128; and cold agglutinins, negative. Lumbar puncture revealed an opening pressure of 480 mm and a closing pressure of 275 mm of water. The cerebrospinal fluid contained 3 WBCs and 1 RBC; all WBCs were mononuclear cells. Glucose was 59 mg/100 ml and protein, 21 mg/100 ml. Blood and CSF cultures revealed no bacterial growth. Skull x-rays revealed sclerotic right mastoid air cells but were otherwise within normal limits. Results of CAT scan with contrast enhancement and isotope brain scan were within normal limits.

Based on past history of both chronic and acute otitis media, and the presence of increased intracranial pressure without an obvious cause, a presumptive diagnosis of lateral sinus thrombosis was made. Selective retrograde bilateral carotid arteriograms revealed thrombosis of the right transverse sinus *(Figure 1)*. The left transverse sinus and superior sagittal sinus were patent.

Because of the patient's excellent clinical condition, we chose to defer surgery and treat her conservatively. After suitable cultures were obtained, she was given chloramphenicol 70 mg/kg/day; phenobarbital, 40 mg/8 hrs; dexamethasone, 8 mg/8 hrs; and glycerol, 1 gm/kg/8 hrs. Within three days of initiation of this therapy, left VI nerve paralysis disappeared. Within eight days, examination revealed a definite decrease in papilledema. Since all cultures were reported as negative for bacterial growth, antibiotic therapy was discontinued after ten days. Two and one-half weeks after onset of vomiting, funduscopic examination yielded normal results. A repeat arteriogram approximately three weeks after onset again revealed a filling defect in the right lateral sinus with excellent filling of the left transverse sinus.

The patient was maintained on fluid restriction and oral glycerol at a dosage of 40 gm three times/day. She was kept on



Figure 1A. Anteroposterior view.

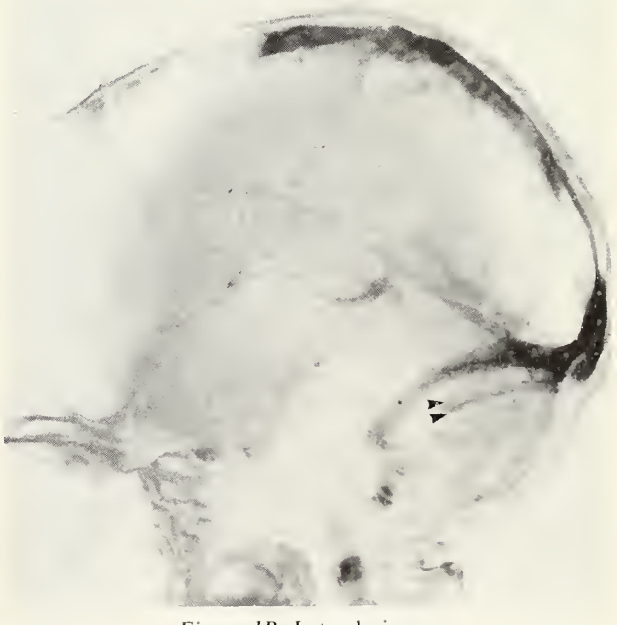


Figure 1B. Lateral view.

Venous phase of right carotid angiogram demonstrating complete occlusion of the right transverse sinus (arrows). The left transverse and sigmoid sinuses are patent.

this regimen for approximately three weeks after discharge, at which time she was completely asymptomatic and results of neurologic examination were normal with no evidence of papilledema. She has remained well.

Comment

Although lateral sinus thrombosis was seen frequently in the pre-antibiotic era, it now is only rarely encountered and thus few recent reports have appeared.

A variety of surgical procedures have been advocated for treatment of this condition. Most sources recommend mastoidectomy as the procedure of choice, possibly with added incision of the thrombosed sinus or ligation of the internal jugular vein. It is significant that almost all cases reported in the literature to date[1-8] have involved patients who have shown more systemic toxicity than our patient, who remained alert and ambulatory with no clinical or laboratory manifestations of systemic toxicity.

In 1967, Greer[9] suggested that some patients with lateral sinus thrombosis might be treated with antibiotic therapy alone. We believe that our patient represents an example of such a situation and recommend that in certain conditions where the patient's course can be carefully monitored, a trial of conservative therapy should be considered.

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References may be obtained from the authors.

EPILEPSIA TORNADO

Epileptic vertigo is a rare type of focal seizure. It has long been known that dizziness may accompany an epileptic seizure, but whether it can, without other features of epilepsy, constitute a seizure is more problematical. Behrman,[1] in an extensive analysis of vertigo and dizziness, came to the conclusion that (1) it may be a postictal vertigo with or without tinnitus; (2) vertigo may be preictal with some evidence of labyrinthine dysfunction between attacks; and (3) there may be a history of interictal disturbance and epileptic fits without warning.

The subject of epileptic vertigo was further discussed by Behrman and Wyke,[2] and they suggest that it can be divided into two groups. In the first, the peripheral receptor stimulation (labyrinth) produces seizure discharges in the vestibular nuclei and then progresses, evolving into an ictus with vertigo and motor phenomenon. In the second group, there is an episode of severe vertigo without any preictal labyrinthine significance, but no progression to motor symptomatology or loss of consciousness. The discharges in the second group emanate from temporal lobe neurones as opposed to the labyrinth in the first group. The case here described, with a review of the literature, belongs to the second group.

Case Report

A 43-year-old male was admitted to the Neurology Service for evaluation of intermittent episodes of vertigo of three to four month's duration. He had a history of a six to eight foot fall a year earlier in which he suffered head trauma but did not lose consciousness. His health was satisfactory until about four months prior to admission when he began having episodes of severe vertigo occurring without aura and lasting for several minutes, followed by frontal headaches. He denied loss of consciousness, but was unable to recall events accurately for a few minutes following these episodes. The episodes occurred two or three times per week but he felt well otherwise. He denied tinnitus, hearing loss, or other neurological problems. Family history was unremarkable, and results of general physical and neurological examinations were normal except for mild obesity. Blood sugar, serum chemistries, and liver enzymes were within normal limits. Electroencephalogram was obtained using Grass Model 8-18, Focal 5-7. Cycles per second slowing in the left temporal leads was noted. A repeat electroencephalogram using nasopharyngeal electrodes revealed spike discharges at PG1 electrode position *(Figure 1)*. An attempt was made to provoke seizure via vestibular stimulation, but failed to produce any spike activity or clinical seizure. Electronystagmography was performed using life Tech 3000-2, and results were within normal limits. Formal audiological testing was also unremarkable. A computerized axial tomography scan revealed normal findings. The patient was started on Tegretol, 200 mg three times/day, and has been followed for eight months without episodes of vertigo or other symptoms.

Discussion

Vertiginous epilepsy, or epilepsia tornado, is a rare type of paroxysmal disorder; on the other hand, vertigo and dizziness from disorders of peripheral end organs are more common. Jackson[3] first described this entity and suggested that it could occur as a result of cerebral dysrhythmia. Since then, several authors[4-10] have tried to analyze vertigo and dizziness occurring in association with generalized seizure disorder such as an aura or in a postictal state. Anatomically, cortical representation of vestibular function is rather diffuse in the temporal lobes. Penfield and Jasper[11] showed experimentally that vertigo may be induced by stimulation of the temporal lobes. Barac[12] was of the opinion that these seizures are usually due to temporal lobe dysfunction, and he expressed doubt that labyrinthine stimulation produces secondary epileptic phenomenon, in contradistinction to the suggestion made by Behrman and Wyke.[2] In the present case, we were unable to produce any dysrhythmia on labyrinthine stimulation. Spike discharges occur independently.

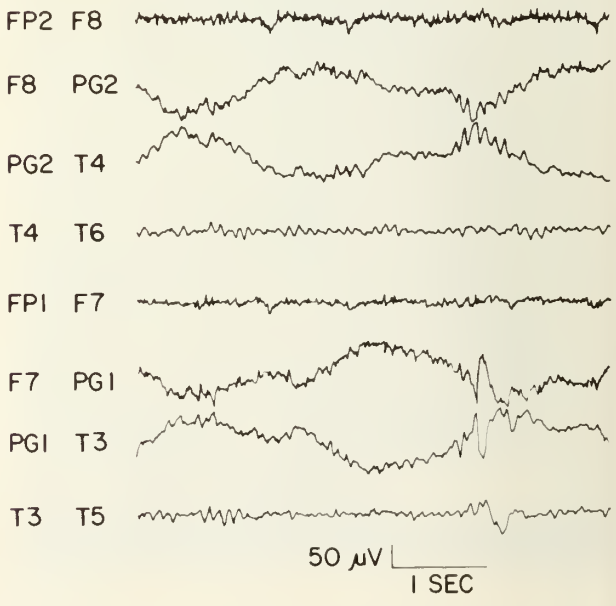


Figure 1. Spike discharge at PG1 position.

Wesseley[13] and Orban *et al.*[14] analyzed cases of epileptic vertigo and found a high incidence of head trauma associated with this type of seizure.

Alper[15] critically reviewed the literature and considered a period of amnesia or loss of consciousness during the episode of vertigo to be one of the primary clinical features differentiating it from peripheral causes. In the present case, the history is consistent with this observation.

Pedersen *et al.*[16] in their analysis of a large number of cases made some important observations. They found the age of onset in this type of cerebral dysrhythmia to be early (none after the age of 60 years), slightly more common in women, and with a history suggestive of other symptoms of temporal lobe dysfunction. They commented on the importance of electroencephalography in this type of paroxysmal discharges. They found dysrhythmia in eight of 14 cases with primary paroxysmal vertigo. Nasopharyngeal electrodes were not used in their series. Since vertiginous epilepsy is a temporal lobe dysrhythmia, nasopharyngeal leads should be more frequently utilized in these cases. For the same reason, Tegretol (carbamazepine) is perhaps the drug of choice for controlling such dysrhythmias.

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References may be obtained from the author.

UNILATERAL PULMONARY EDEMA IN SWYER-JAMES SYNDROME

Unilateral pulmonary edema is rare but when it does occur may present a diagnostic problem. Calenoff *et al.*[1] have identified at least 18 clinical situations that have been reported to be associated with unilateral pulmonary edema. We report a case of unilateral pulmonary edema in a patient with Swyer-James syndrome.[2] Development of unilateral pulmonary edema during the hospital stay raised this possibility. Subsequent investigation confirmed the diagnosis.

Case Report

A 79-year-old white male was admitted to the hospital for increasing shortness of breath. At age 42 he had experienced a severe attack of pneumonitis involving the left lung. He gave a 60 pack year history of cigarette smoking with several admissions to the hospital for exacerbation of chronic obstructive lung disease. On the fourth hospital day, the patient became acutely ill with shortness of breath, cyanosis, and frothy sputum. Physical examination disclosed ronchi and rales in the right lung and decreased air entry in the left lung. S_3 gallop was audible. Chest x-ray showed unilateral pulmonary edema of the right lung with fluid in the minor fissure of the left lung (*Figures 1* and *2*). He was intubated and given intravenous digoxin and furosemide resulting in improvement in the clinical symptoms and clearance of pulmonary edema on chest x-ray obtained four hours later *(Figure 3)*. Serial electrocardiograms and enzyme estimations revealed acute anterior wall myocardial infarction.

After the patient recovered from the acute episode a ventilation lung scan, performed following inhalation of ^{133}xenon in gas demonstrated decreased air entry but a normal washout from the left lung. There was xenon retention in the right lung base. Perfusion lung scan performed following intravenous administration of 99m technetium labeled microspheres showed almost total absence of perfusion to the left lung with perfusion defects in the right lower lobe. Pulmonary angiography showed a diminutive left pulmonary artery with diminished peripheral vascular branches *(Figure 4)*. Bronchogram showed minimal bronchiectatic changes predominantly involving posterior basi-

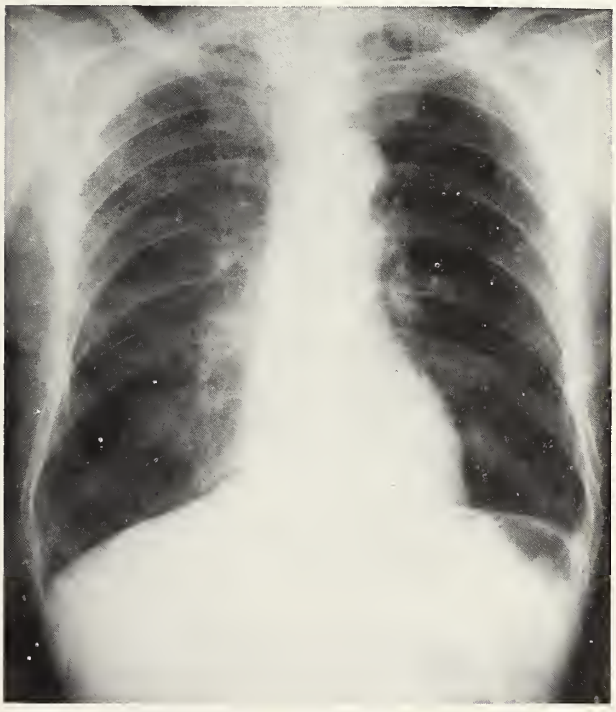


Figure 1. Admission chest x-ray demonstrating clear lung fields with minimal hyperlucency of the left lung.

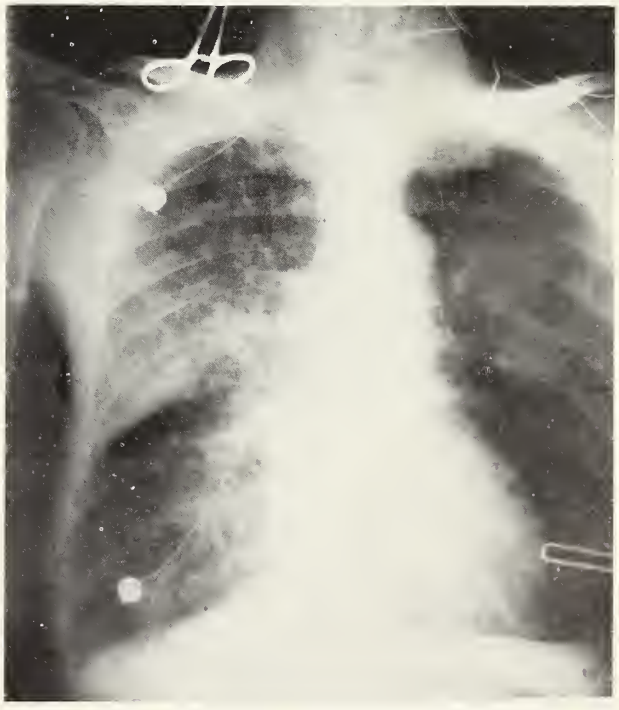


Figure 2. Chest x-ray taken during acute pulmonary edema showing unilateral right sided pulmonary edema with fluid in the short fissure of the left lung.



Figure 3. Repeat chest x-ray obtained four hours following appropriate treatment showing improvement in pulmonary edema.

lar segment. Pulmonary function studies demonstrated findings consistent with severe obstructive lung disease. Chest x-ray in expiration did not show a mediastinal shift.

Discussion

Unilateral hyperlucent lung syndrome was first described by Swyer and James in 1953.[3] In 1954, Macleod reported nine additional patients with this syndrome.[4] Since then there have been several additional reports of this syndrome in children and adults. Clinical features are variable and may include repeated respiratory infection or dyspnea, or patients may be totally asymptomatic. Radiographic findings include normal or decreased lung size, decreased lung markings, and a smaller hilar shadow when compared to the opposite lung. A chest x-ray during expiration demonstrates mediastinal shift to the opposite side indicative of air trapping in the affected lung.[4]

Bronchogram of the involved lung demonstrates mildly dilated smaller bronchi with a beaded appearance. The ends of some of these bronchi are clubbed. There is a lack of alveolar filling.[5] Pulmonary function studies show obstructive airway disease.[5] There is delayed xenon washout on ventilation study in the affected lung with markedly diminished perfusion on perfusion lung scan.[6] Pulmonary angiography reveals a markedly diminished pulmonary artery on the affected side with diminished peripheral vascular branches.[7]

The patient presented in this case report had Swyer-James syndrome involving the left lung. This was most likely brought on by an episode of severe pneumonia 37 years earlier. The unusual feature in this patient was the lack of significant air trapping in the affected lung as demonstrated by a lack of mediastinal shift on expiration and a normal washout of xenon from the left lung. The bronchogram showed minimal bronchiectatic changes primarily involving the left posterior basal



Figure 4. Pulmonary angiogram showing small left pulmonary artery with decreased peripheral branches.

segment. However, the lack of significant perfusion to the left lung on the lung scan is quite striking. This suggests that vascular changes are more advanced and more pronounced in this patient.

Pulmonary edema was precipitated by an acute anterior wall infarction on the fourth hospital day. Rapid clinical response to digitalis and diuretics with improvement demonstrated by chest x-ray obtained four hours later supports this diagnosis. Occurrence of unilateral pulmonary edema in this patient prompted further diagnostic workup which established Swyer-James syndrome.

Unilateral pulmonary edema is rare, but when present, may be confused with unilateral interstitial or alveolar infiltrate. Awareness of this entity is necessary to ensure proper treatment.

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TEMPORARY RESISTANCE TO HEPARIN AND WARFARIN IN TREATMENT OF DEEP VEIN THROMBOSIS

The drug of choice for treatment of deep vein thrombosis (DVT) is Heparin. Due to its immediate onset of action and relative safety, it is usually started as soon as diagnosis is made, and it is administered most efficiently by continuous intravenous drip in dosages of 24,000-40,000 units/day.[1] With the recent advent of the thrombolytic agents streptokinase and urokinase, experience in clot lysis *in vivo* is being obtained, but Heparin continues to be the first line drug, usually followed by the oral administration of warfarin. It has been reported[2] that extensive DVT or phlebitis causes a resistance to Heparin, but recent literature does not mention it as a frequent occurrence. This case report involves a young patient with typical DVT who did not respond to Heparin

administered alone or in conjunction with warfarin so that treatment with streptokinase was mandatory.

Case Report

A 37-year-old male sustained a closed fracture of the left first metatarsal bone; four weeks later he developed massive swelling of the entire left leg with pain and inability to walk.

Physical examination on admission to the hospital revealed typical features of DVT. There was slight discoloration of the skin of the distal foot where the injury had occurred. Pitting edema of the left lower extremity was evident, and swelling in the thigh was so massive that the femoral arterial pulsations were not easily felt. The patient had great difficulty in bending the left knee joint. There was no previous history of phlebitis, thrombosis or embolism, and no family history of these afflictions. He had experienced two minor episodes of chest pain a week prior to admission to the hospital.

Laboratory data revealed normal blood count with platelets, and a mildly elevated fasting blood sugar on the chemistry profile. Chest x-ray results were negative. The admission prothrombin time and partial thromboplastin time were both within normal limits, below 12.5 and 35.0 seconds respectively. The antithrombin III (AT III) was in the lower limits of normal range (81-120%) at about 88 per cent. Perfusion lung scan showed multiple defects in the right lung indicative of emboli. Subsequent scan showed infarction, and later during hospitalization, the patient developed right pleural effusion thought to be secondary to the pulmonary infarctions.

Immediately following diagnosis of DVT, therapy was started with intravenous Heparin using a loading dosage of 3,000 units followed by continuous drip of 1,000 units/hr by pump infusion. With no response to this dosage after 24 hours, Heparin was gradually increased to 1,200 and subsequently to 1,500 units/hr, and the partial thromboplastin time (PTT) was closely monitored. With no change in PTT and no clinical improvement by the fourth day of this treatment, warfarin was added at a dosage of 15 mg/day. However, after 48 hours no change was noted in PTT or prothrombin time. With continuing pulmonary emboli and deteriorating condition, it was elected to stop Heparin and warfarin and administer streptokinase intravenously, using a loading dosage of 250,000 units followed by 100,000 units/hr continuously. This produced a good clinical response with the swelling of the leg diminishing. PTT increased from an earlier 22-27 seconds to 34 seconds. Prothrombin time also increased to 16.5 seconds from an earlier reading of 12.5 seconds.

Following 40 hours of treatment, streptokinase was terminated and Heparin was restarted at 1,000 units/hr combined with warfarin, 10 mg/day. However, there was no response in PTT, which had come down to 23.5 seconds following 24 hours of this regimen, so Heparin was stopped and warfarin alone was continued in an effort to elevate prothrombin time above the previously noted 16.5 seconds. Again there was no response to warfarin, and the patient began developing phlebitis in the right arm. This forced the use of streptokinase for the second time, and it was again administered for approximately two days in an effort to prevent massive pulmonary emboli and to improve the condition of the leg. Streptokinase had to be terminated due to severe phlebitis at the site of infusion with subsequent edema of both arms and great discomfort. Streptokinase was followed by Heparin subcutaneously, 8,000 units/six hrs, combined with warfarin, 15 mg two times/day. Following 60 hours of this treatment, PTT rose to 43 seconds and prothrombin time to 25 seconds. Heparin was then discontinued, and the patient was maintained on warfarin, 15 mg/day, with the prothrombin time stable at around 20 seconds. Meanwhile, the patient's general condition improved, edema of the leg totally disappeared, and there was about 90 per cent improvement of the edema of both upper extremities. There were no chest pains or other clinical signs of fresh pulmonary emboli, and the patient was not tachypneic. He was sent home on warfarin, 15 mg/day, fully ambulatory and in no pain.

Conclusion

This case illustrates the fact that massive DVT can itself cause temporary resistance to Heparin, and perhaps resistance to warfarin as well. At a time when anticoagulation should be pursued vigorously, the patient cannot be undertreated as there is a grave risk of pulmonary emboli; it is therefore essential to judiciously increase the Heparin dosage to a maximum toleration — perhaps 40,000 units/day — to keep PTT around one and one-half to two times normal. If the patient gives a previous history of DVT or a family history of this condition, AT III level should be obtained, as a deficiency of this protein — previously known as Heparin cofactor — will forewarn of poor response to Heparin.[3, 4] Warfarin would then be indicated as it does not depend on AT III for its function. This particular patient had a mild depression of AT III, and as there was a negative family history, it is more likely that the patient's deficiency was related to the massive venous thrombosis itself.[5] Streptokinase is a very potent thrombolytic agent, and its use should be reserved for massive pulmonary emboli or DVT when duration is four to seven days.[6] The chances of local phlebitis at the site of infusion with streptokinase should also be considered, as this can lead to difficult problems in maintaining continuous intravenous drug therapy.

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References may be obtained from the author.

THE SYNDROME OF INAPPROPRIATE SECRETION OF ANTIDIURETIC HORMONE ASSOCIATED WITH AMITRIPTYLINE OR TRIFLUOPERAZINE ADMINISTRATION

For clinical purposes, parameters of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) may be defined as: (1) no clinical evidence of intravascular volume depletion or edema; (2) normal renal function; (3) normal adrenal function; (4) simultaneous hyponatremia and hypo-osmolality of the serum; (5) inappropriate hyperosmolality of the urine for the simultaneous tonicity of the plasma; and (6) inappropriate urinary sodium excretion concomitant with the serum sodium concentration.[1] SIADH has been associated with a variety of clinical disorders; the most frequent of which is bronchogenic carcinoma, particularly small cell or oat cell.[2] In addition, an increasing number of drugs have been implicated in SIADH. This report presents a case of SIADH in a patient receiving both amitriptyline (Elavil) and trifluoperazine (Stelazine).

Case Report

A 52-year-old female presented to Truman Medical Center with a one-day history of nausea and vomiting. Past medical history revealed a prolonged period of "nervousness" for which she was receiving amitriptyline (Elavil), 50 mg at bedtime, and trifluoperazine (Stelazine), 5 mg three times/day. Physical examination was unremarkable except for marked lethargy. There was no evidence of intravascular volume depletion, edema, or adrenal insufficiency. Pertinent laboratory values included serum sodium, 114 mEq/L (normal 135-148 mEq/L); serum potassium, 3.8 mEq/L (normal 3.5-5.3 mEq/L); serum chloride, 79 mEq/L (normal 96-107 mEq/L); serum bicarbonate, 21 mg/dl (normal 7-22 mg/dl); and serum creatinine, 0.6 mg/dl (normal 0.5-1.0 mg/dl). Serum glucose was 127 mg/dl. Initial serum

TABLE I

	Day 1	*Day 2*	*Day 3*	*Day 5*
Serum sodium (mEq/L)	114	121	137	138
Serum osmolality (mOsm/kg H_2O)	246		260	279
Urine osmolality (mOsm/kg H_2O)	245			
Urine sodium (mEq/L)	6			

osmolality (with a simultaneous serum sodium being 117 mEq/L) was 246 mOsm/kg H_2O (normal 275-300 mOsm/kg H_2O). Urine osmolality was 245 mOsm/kg H_2O, and urine sodium, 6 mEq/L. Thyroid function tests yielded normal results. Other laboratory results were unremarkable. A flow chart of laboratory values during the patient's hospitalization is provided in *Table I*. A diagnosis of SIADH was made for which treatment consisted of water restriction and discontinuance of medications. Recovery was uneventful, and the patient was not rechallenged with either medication. Water intake was unrestricted upon discharge.

Discussion

SIADH has been recognized with increased frequency, particularly cases associated with drug administration. The list of drugs capable of inducing SIADH includes oral hypoglycemic agents (chlorpropamide), anti-neoplastic agents (cyclophosphamide), and thiazide diuretics.[3-5] Amitriptyline has been infrequently associated with SIADH; trifluoperazine has not been reported as a causative agent.[6] Our case depicts two aspects of SIADH that deserve further discussion. The first is the lower initial urine osmolality than serum osmolality. It is commonly thought that the reverse must be true in SIADH. Although osmolality of the urine usually is above that of serum in SIADH, all that is required for the development of SIADH is that the urine be less than maximally dilute in the face of hypotonicity of the serum.[7] In other words our patient's urine should have been more dilute in the face of her serum hypotonicity. Thus she was retaining free water resulting in hyponatremia and the signs and symptoms of water intoxication. The second point of interest is her low urine sodium excretion. Typically patients with SIADH excrete a relatively high sodium concentration. However, this high loss of urinary sodium usually occurs during the induction phase of SIADH. Eventually patients with SIADH reach a new steady state in urinary sodium excretion at which time urine loss will match sodium intake. Our patient's intake was very low during the time prior to admission and early hospitalization. How amitriptyline or trifluoperazine produces SIADH is unknown. Amitriptyline has significant anticholinergic side effects which may result in mouth dryness. Lewis has suggested that hyponatremia may be produced by excessive water intake to combat mouth dryness while taking amitriptyline.[8] The history indicated our patient's fluid intake was not excessive. Although adrenal function studies were not obtained, clinical and laboratory evidence, in addition to an uneventful recovery while off medication, make adrenal disease highly unlikely. Many persons are being treated with amitriptyline or trifluoperazine. In most instances changes in behavior while on these drugs is attributed to the underlying psychiatric illness. This report suggests that SIADH indeed occurs with amitriptyline or trifluoperazine and that significant changes in behavior while on these medications should not automatically be diagnosed as of psychiatric origin. Measurement of serum sodium would be an effective screening test for SIADH in these individuals.

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Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Hypothyroidism and Coma

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Severe primary hypothyroidism in the adult is a serious medical problem. It is associated with an acceleration of atherosclerosis, diminished functional ability of the patient, and a number of associated medical problems. When unrecognized, the chronically hypothyroid patient is at risk for the development of myxedema coma. This life threatening medical emergency carries a high rate of mortality and should be avoided by proper recognition and treatment of hypothyroidism during its earlier stages.

Clinical Features

Thyroid hormone deficiency affects virtually every organ in the body. The clinical picture of hypothyroidism depends upon a variety of factors including the rate of onset of the deficiency and its severity; it is not unusual for gross deficiency of the thyroid hormones initially to be associated with minor clinical features. In time the more classical findings associated with hypothyroidism become manifest. Overt hypothyroidism (myxedema) is generally associated with a spectrum of clinical features which have been well described for over 100 years.

Skin Changes: Dry and flaking skin with hyperkeratosis over the flexures characterizes hypothyroidism. In addition, the skin feels cool to the touch and takes on a faint yellow tinge due to hypercarotenemia associated with impaired conversion of carotene to vitamin A. The coarsened features of the skin are accompanied by subcutaneous swelling most obvious in the eyelids and periorbital tissues and the dorsum of the hands and feet. This accumulation of fluid and hyaluronic acid is responsible for the term myxedema or mucinous edema. The hair becomes coarse and dry with a peculiar thinning of the outer thirds of the eyebrows frequently observed.

Nervous System: In severe hypothyroidism a slowing of intellectual functions including speech frequently occurs, although in some instances the mental alertness of the patient is well preserved. This combination of slow, deliberate speech with unimpaired mental function gives rise to the term "myxedema wit." Mucinous edema of the vocal cords frequently causes coarse, raspy, or hoarse speech. Peripheral neuropathies, perceptive hearing loss, and vertigo all result from these effusions. The latter may be so pronounced as to be confused with cerebral ataxia.

Muscle and Joints: Hypothyroidism may present primarily as a rheumatic syndrome since generalized aches and pains in muscles and joints are frequent. A characteristic and specific finding of hypothyroidism is a prolonged relaxation phase of the tendon reflexes despite a brisk or normal contraction phase best observed by gentle percussion of the achilles or biceps tendons.

Alimentary System: Decreased peristalsis and constipation are common. Acute ileus mimicking a surgical emergency is a rare complication. This may result from either myxedematous deposits in the bowel wall or a peripheral autonomic neuropathy. A complication of hypothyroidism observed in two cases by the author is ascites associated with pleural and pericardial effusions responding rapidly to thyroid hormone therapy.

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Cardiovascular System: In hypothyroidism cardiac output is reduced as a result of both decreased stroke volume and heart rate. Pericardial effusion is frequent even when cardiac enlargement is not pronounced on the chest x-ray. It appears that pericardial effusion accounts for most of the findings associated with "myxedema heart disease," *i.e.* cardiac enlargement and low voltage on the electrocardiogram. The effusion and associated findings rapidly improve with thyroid hormone replacement.

An increased prevalence of ischemic heart disease is now well established in patients with hypothyroidism. There is a recognized association between hyperlipoproteinemia, hypercholesterolemia, and hypothyroidism. Although the serum lipoprotein abnormalities may improve with treatment, the presence of coronary artery disease poses therapeutic problems since angina pectoris may be precipitated or aggravated by thyroid hormone replacement.

Hemopoietic System: Approximately 25 per cent of patients with hypothyroidism have anemia. Although pernicious anemia may co-exist with hypothyroidism and account for a megaloblastic anemia, the majority of patients experience normochromic, normocytic anemia due to lack of thyroid hormone. Such patients will not respond to hematinic agents such as folate, iron or B^{12}, but anemia will be rapidly corrected with thyroid hormone therapy.

Myxedema Coma

Myxedema coma is the end stage of neglected or undiagnosed hypothyroidism. It is characterized by coma, hypothermia, cardiovascular collapse, hypoventilation and severe metabolic derangements, including hyponatremia, hypoglycemia, and lactic acidosis. Myxedema coma must be distinguished from the mild to moderate hypothyroidism associated with lethargy, sleepiness, and cold intolerance. Such hypothyroidism is not myxedema coma and should not be treated as such.

Causes: Generally myxedema coma requires several years to develop. It usually occurs in elderly people who have been without medical care, but occasionally develops after ill-advised administration of sedatives or other psychotropic drugs (the half-life of which is prolonged) to patients with unrecognized hypothyroidism. Coma is rare but the frequency is increasing probably because of increased hypothyroidism secondary to I^{131} therapy for thyrotoxicosis. Myxedema coma is rarely reported in patients under the age of 50 years; advanced age has significance with respect to high mortality rates associated with this condition. The majority of patients in one series presented with coma during the winter months, suggesting exposure to cold may be a precipitating factor.

Associated Complications: The state of coma may not be directly related to thyroid deficiency *per se;* in most instances, complications resulting from the hypothyroid state can account for the comatose state. Carbon dioxide retention is frequently present due to changes in the thoracic ventilatory apparatus and the respiratory center, as well as compromised respiration due to pleural effusions and ascites.

Hyponatremia often complicates the condition of the patient with myxedema and coma. These patients have markedly impaired excretion of water loads. Thus the hyponatremia is dilutional and not the result of chronic sodium wasting. Water diuresis is one of the first observed responses to thyroid hormone therapy in severe myxedema.

Hypothermia occurs in more than 80 per cent of patients with myxedema coma. This may be underestimated since many thermometers do not register below 34C and temperatures as low as 24C have been reported. Temperatures below 32C have been associated with a very grave prognosis. If the temperature is above 36C, an associated infectious process should be suspected. This additional complication may be the precipitating cause of the coma, particularly if pulmonary infection contributes to hypoventilation.

There is controversy concerning the function of the other endocrine glands in severe myxedema. Reduction of cortisol turnover rate reduces urinary excretion of corticosteroids; however, plasma cortisol level is generally normal. Although the pituitary-adrenal axis responds to stimuli such as vasopressin, hypoglycemia and metyrapone, the response to stress is often impaired. Myxedema coma rarely occurs as the result of pituitary hypothyroidism. In even the most severe and protracted cases of primary hypothyroidism with coma the pituitary thyrotrophs continue to secrete thyrotropin. Hyperplasia and even adenoma formation of these anterior pituitary cells may result from long-standing hypothyroidism.

Treatment of Myxedema Coma

The prognosis is guarded in myxedema coma with greater than 50 per cent mortality. Successful treatment depends upon prompt recognition of the clinical signs of hypothyroidism in the comatose patient. Since undue delay will jeopardize the chance for recovery, there is no justification for withholding therapy until laboratory confirmation of hypothyroidism is obtained. Serum should be obtained for

measurement of the thyroxine (T_4) and pituitary thyrotropin (hTSH) concentrations, but therapy should be promptly instituted once the clinical diagnosis is made.

Administration of Thyroid Hormone: The rational course of therapy is the delivery of thyroid hormone. However, myxedematous patients are frequently extremely sensitive to the metabolic effects of the thyroid hormone and may sustain cardiac damage. Because of this concern there was reluctance in the past to administer adequate doses of thyroid hormone to the patient with myxedema coma. There is no need for this concern because clinical studies in which large doses of sodium l-thyroxine were given intravenously have shown good results. Liothyronine (T_3) has been used in the treatment of myxedema coma; however, a parenteral preparation of T_3 is not available, and there is no evidence of responses superior to those obtained from intravenous thyroxine. A currently accepted therapeutic approach for the administration of thyroid hormone to the patient in myxedema coma is an initial dose of 200-400 μg sodium levothyroxine intravenously followed by 100 μg intravenously daily until oral therapy can be given. Before each dose the patient should be carefully examined for signs of hypermetabolism or cardiovascular decompensation. When the condition has stabilized and oral T_4 therapy has been started, levothyroxine should be given in small daily doses (0.025-0.05 mg) and increased slowly as in uncomplicated hypothyroidism.

Ventilatory Assistance: Frequent hypoventilation resulting in CO_2 retention, hypoxia, and respiratory acidosis necessitates maintenance of adequate ventilation and oxygenation. Arterial blood gases should be measured and ventilatory assistance provided if necessary. Sedatives or drugs that depress respiration should be avoided and pulmonary infections should be vigorously treated.

Adrenal Hormone Replacement: Evidence for routine steroid therapy is not clearly established, but ACTH response to stress in the hypothyroid patient may be impaired. In addition, primary hypothyroidism may co-exist with primary adrenal insufficiency (Schmidt's syndrome). Supportive therapy with steroids must therefore be recommended. Hydrocortisone, 100-300 mg daily by infusion, is required in the early stages. This can be replaced by oral therapy and gradually tapered off as clinical recovery occurs.

Hyponatremia: Dilutional hyponatremia seen in myxedema coma may produce severe enough reduction in serum osmolality to induce a generalized seizure. If a spinal tap is performed, both spinal fluid pressure and protein concentration may be found to be increased. Recognition that these alterations occur as a result of the hypothyroid state is important from a diagnostic standpoint. If the serum sodium is extremely low (below 115mEq), it may be advisable to administer a small amount of hypertonic saline. Correction of serum sodium to above 120mEq is unnecessary and may further compromise the cardiac status of the patient who is already volume expanded. At this point dilutional hyponatremia can be treated by a trial of fluid restriction with monitoring of serum electrolytes.

Hypothermia: Covering with blankets to conserve body heat loss is adequate for most patients. Active warming should be avoided since it increases the caloric requirements of the body surface and may induce peripheral vasodilation thus aggravating hypotension.

Supportive Measures: In severe vascular collapse and hypotension, it may be necessary to administer

TABLE I
SUMMARY OF MYXEDEMA COMA

Causes

1. A severe, "end stage" form of primary hypothyroidism from:
 a. Hashimoto's (chronic) thyroiditis
 b. Radioactive iodine therapy
 c. Thyroidectomy
2. May be precipitated by drugs (sedatives, narcotics, phenothiazines), stressful hospital procedures, and infections.
3. Rarely seen in young individuals or in association with pituitary hypothyroidism.

Clinical features

1. Elderly patients
2. Dry skin, puffy edema
3. Abdominal distention, fecal impaction
4. Delayed return of deep tendon reflexes
5. Hypothermia
6. Hyponatremia
7. EKG changes of low voltage and bradycardia
8. Hypercholesterolemia
9. Pericardial and pleural effusions
10. Respiratory acidosis due to CO_2 retention

Management

1. Draw serum thyroxine and TSH
2. Give 200 μg sodium levothyroxine intravenously
3. Assist ventilation
4. Give 200 mg hydrocortisone intravenously during 24 hrs
5. Administer water restriction and hypertonic saline for severe hyponatremia
6. Give glucose and pressor drugs as needed
7. Do not actively warm the patient
8. Treat associated illness and avoid sedatives

pressor agents; however, once thyroid hormone is given, increased sensitivity develops to these drugs. Therefore, the patient must be closely monitored and the drug dose adjusted accordingly. Hypoglycemia is occasionally significant and should be treated with parenteral glucose solutions in small volumes so as to avoid fluid overload.

Summary

Severe hypothyroidism leading to coma represents a true medical emergency requiring prompt and aggressive treatment. Elaborate testing is unnecessary because the diagnosis can be established by clinical signs and symptoms. Myxedema coma represents not only a failure of the body to adjust to long standing hypothyroidism, but in a larger sense, a failure of society to recognize and treat the hypothyroid patient. More awareness of the subtle clinical features and more refined corroborative laboratory tests should lead to earlier diagnosis and treatment of hypothyroidism and reduce the frequency of coma. A greater need is the establishment of physician-patient rapport to insure the continuing thyroid therapy throughout the lifetime of the hypothyroid patient.

Self Assessment Questions

1. Hypothyroidism has not been shown to be associated with an acceleration of atherosclerosis. (T or F)
2. Thyroid hormone deficiency state is irreversible in the adult. (T or F)
3. A characteristic and specific finding of hypothyroidism is a prolonged relaxation phase of the deep tendon reflexes. (T or F)
4. Myxedema coma should be distinguished from moderate hypothyroidism associated with lethargy and cold intolerance. (T or F)
5. The treatment of choice of myxedema coma is liothyronine (T_3). (T or F)

(Answers on page 477)

The Smallpox Story

(Continued from page 456)

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The President's Message

I'll Be There!

Will you be there? In a democracy there are few things much more important than participation in the political process. While this participation for some may include running for office and serving when elected, most of us must support the candidacy or "platform" of others. It is indeed unfortunate if we fail to participate even to that limited extent.

Involvement best starts with the encouragement of qualified persons to enter the race and, once the candidates are chosen, to support the candidate of your choice with vigor. We can all provide support not only with money but with expressions of our support publicly to friends and patients. Although such personal support is invaluable, support of KaMPAC serves another important and unique function.

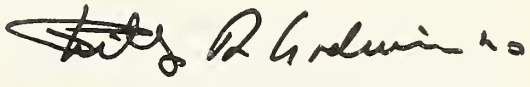
KaMPAC is an organization made up of our colleagues who evaluate races on the state and national levels and contribute to candidates who are judged most supportive of medicine's collective point of view. It has a good record of helping elect such persons to office and carries out a function we cannot hope to accomplish on an individual level. I hope you are already supporting individual candidates and KaMPAC. If not, it is still not too late to start.

But the political process depends on more. The AMA is sponsoring a campaign called "I'll be there!" It is designed to encourage us all to see that we are registered and turn out to vote. Ask your colleagues if they are going to vote. Encourage them to do so. Remind your office staff to vote and give them adequate time off on election day if needed. Encourage 100 per cent participation by your county society members and their spouses. Members who will be out of town can vote with absentee ballots.



When it comes time to vote, let's all be there. I'll be there . . . will you?

Fraternally,



President



Sweeter Manners, Purer Laws

Mr. Tennyson probably realized his words would at times be stolen out of context by lesser mortals to make a point but he could hardly have foreseen their present use. In this case, they introduce the fact that the November issue of the *Journal* will be devoted to medicolegal subjects, and our comments may be considered prefatory. Although medicolegal matters have had much exposure in medical publications in recent years, these will have, we think, an attraction for our readers since the lawyer-contributors are based in Kansas and have consequent familiarity with the local professional relationships. Some have gained particular insight into medical practice by provision of legal services to medical organizations and individuals, and some of our own members who hold degrees in both medicine and law are included.

As intellectual disciplines usually classed in the higher order, medicine and law have some features in common, but through the years communication has been hampered by the very real differences. As the regulator and arbiter of social order, law seeks to establish and apply the controls of society on the individual person or group. Its concern with individuals as individuals is still dictated by those controls. Medicine, on the other hand, directs its concern primarily toward the individual and secondarily to society by outward extension from that individual. It is unfortunate but not surprising, then, that when the two meet it is often across this individual when the service of the physician is considered remiss. While the situation may have developed as a matter of individual effect, it is within the framework of legal definition and social expectation that the culpability of the physician is measured. This is not to ignore other areas of medical-legal contact but this is quite sufficient to make joyful comradeship hard to come by.

Consideration of the relationship of these two primary service entities requires some perspective. The world is struggling with transitions in all dimensions. The almost total elimination of time-space barriers is usually referred to as opening new horizons. As a matter of fact, by compelling us to look at the finitude of the planet, it compels us to work within increasingly limited horizons. Much that is interpreted as cultural progress is a phenomenon of vertical displacement (both actual and figurative) replacing the horizontal expansion which began with the age of exploration. In the sociopolitical context, the "deprived" are now "emerging" and demanding an equivalence they cannot provide for themselves or handle if they could. This is no derogation of their potential, but the efforts of the social and political zealots to bring all to a common social denominator immediately call for a panoply of social changes more rapidly than the participants can assimilate them.

Medical resources, while trying to cope with the intrinsic demands imposed by professional progress, are increasingly confronted with extrinsic demands to meet the anticipated "new order." The consequent infringement on medical territory and prerogative is promoted by the protagonists as an expression of freedom. It is proclaimed as the release of the patient from the strictures of the system, which grew from its origin as a basic struggle for survival to a technologic exercise directed so unrelentingly toward method it has lost contact with the patient.

So, in the world of "health" — in this instance, we include the total inventory: disease, injury, therapy, method, personnel, recompense — this is producing its own form of revolution. As in the world at large, the charges and countercharges that are mounted by the proponents of various ideologies refuse to stay within well-defined boundaries. The current demands for regulation and control of medical and hospital practice, for example, are not primarily professional but fiscal and social. (Perhaps

this explains the fascination and approval the public extends toward medical activities and progress while rebelling against the financial requirements of obtaining and applying them.) As payment for medical services is spread across the population (some 90% reportedly paid by insurance or the government, a seemingly legitimate application of the insurance principle), those charged with providing those services are increasingly subjected to the threat and fact of both professional and fiscal policy. In the course of this social readjustment, the recipients of service or substance are "freed" at a rate equivalent to the rate at which the providers are subjected to regulations and controls.

Not surprisingly, this upheaval of transition is accompanied by a changing relationship between medicine and law. This is not to say it has had an effect of unidirectional cohesion — rather some changes in attitude as well as recognition by the medical profession of the need to be more conversant with the function of law. There is a great deal more communication between the two professions. Though there are many areas of mutual concern, the problem of medical malpractice would undoubtedly be the first to come to mind when the word, medicolegal, is used. Distressing as the recent history of medical malpractice litigation has been, it has served to remind physicians that for every plaintiff's lawyer there is a defendant's lawyer. This may not go far toward making a malpractice suit palatable to the subject physician, but it does serve to increase the exposure of each to the other with the awareness that the destinies of the two are becoming more entwined, and more benefits can come from objective and dispassionate exchanges than vituperation and hostility.

So, in one way or another, and whatever the benefits of "progress," the method has come to mean regulation and control, and regulations and controls are based in law. Directly or indirectly, permissive or prohibitive, they arrive by legislative action. Transformed to statute, their intent, application, and effect are subjected to judicial test. Their viability is determined by their conformance to legal pattern. Function under them is scrutinized by legal agents. Infractions are charged and tried under law, and exoneration or punishment follow the code established by law.

Every step in the process has numerous ramifications and characteristics complicating its application, and consequently legal guidance is as essential for survival of the medical profession as, for example, sustained medical refurbishing (which, in its current form, is a prime example of the extended social influence). The days when a physician could appear at the forum and cite some medical truisms in support of a worth-while legislative proposal are past. The construction of such proposals must be accomplished with legal expertise anticipating efforts to counter it. Physician testimony must recognize and utilize a high level of legal sustenance to withstand the valid scrutiny of the day.

The Kansas Medical Society operates on the principle that the more this legal guidance can be developed in collective action, the better protected the individual physician (and, consequently, the patient) will be as s/he attempts to provide medical service in a compelling environment of social demands. These papers will be offered, then, not with the expectation of giving final answers or providing permanent solutions — certainly not to give any cook book advice on legal matters — but of enhancing the contacts and dialog necessary to the support of the primary medical concern and obligation, patient care. — *D.E.G.*

Hypothyroidism and Coma

(Continued from page 474)

Answers

1. *False*
2. *False*
3. *True*
4. *True*
5. *False*

Suggested Readings

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AMA House of Delegates

Summary of Actions taken at the 1980 Annual Meeting, Chicago

With 279 delegates, this was the largest House in the history of the Association. The Kansas Medical Society was officially represented by Phillip A. Godwin, M.D., President; two delegates and their alternates; Jerry Slaughter, and Val Braun. Also present from Kansas was William J. Reals, M.D., Wichita, representing the American College of Pathology.

The House considered 249 resolutions and reports pertaining to virtually every interest and concern of the practicing physician. Because of space limitations, only those matters that are believed to be of utmost interest to Kansas physicians are reported here. If you would like to have the details of other discussions, please get in touch with one of your delegates listed below.

Although the American College of Surgeons had withdrawn its representation, the House voted to keep the seat open in the hope that ACS would reconsider. In another action, the House voted to terminate representation from the Canal Zone since it no longer meets the criteria for a constituent society.

There will be no dues increase for 1981 (AMA dues currently stand at $250). Inflation is continuing to exert a damaging effect on the Association and has cost the AMA at least $12 million during the last few years. Since membership dues will continue to be the basic source of revenue for the Association, the only realistic alternative for increasing revenues lies in keeping the present members and bringing in new members. The House of Delegates adopted a report encouraging state and county societies to set AMA membership goals for the coming year.

Principles of Medical Ethics

After two years of intense study and consultation with medical societies and individuals, the Ad Hoc Committee submitted a revision consisting of a preamble and seven sections. The House adopted the revised version by more than a two-thirds vote. The principles were last revised in 1957. The new version reads as follows:

Preamble: The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professionals, and to self. The following Principles adopted by the American Medical Association are not laws, but standards of conduct which define the essentials of honorable behavior for the physician.

I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.

II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception.

III. A physician shall respect the law and also recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.

IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.

V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.

VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environment in which to provide medical services.

VII. A physician shall recognize a responsibility to participate in activities contributing to an improved community.

Joint Commission on Accreditation of Hospitals

The House directed some severe criticism at the JCAH and its survey procedures. Several resolutions expressed deep dissatisfaction with JCAH requirements and the conduct of surveys. A Board of Trustees report said that the JCAH Accreditation Manual for Hospitals tends to prescribe methods of organizing the hospital and the medical staff, rather than concentrating on the quality and excellence of patient care. The House directed the Board to initiate a series of changes for improving the hospital accreditation process.



Alex C. Scott, M.D., Junction City



Lew W. Purinton, M.D., Wichita

Medical Education Accreditation

The 1979 House voted to withdraw from the LCCME. In a mood of conciliation and cooperation, the 1980 House authorized the Board of Trustees to eliminate dual accreditation by the LCCME and the AMA. The House adopted a report outlining proposed changes and accrediting medical education at all levels through ongoing discussions with the parent organizations of the Coordinating Council on Medical Education and the liaison committees. The Board's report said that accreditation of graduate medical education should be modified to make residency review committees responsible for actual accrediting decisions. The Liaison Committee on Graduate Medical Education functions would then change to policy development and appeals. No changes would be needed in the accreditation of undergraduate education. The House also called for continued support of voluntary CME, and asked state associations and specialty societies to support a moratorium on additional mandatory CME and mandatory recertification programs.

Health Maintenance Organizations

The House acted to continue its policy of neutrality and fair market competition among all systems of health care delivery. It also reaffirmed a policy against preferential subsidies to any one form of health care delivery and protested the use of federal tax money for advertising to promote HMOs.

National Health Insurance

The AMA will continue to advocate in a positive manner the superiority of a voluntary, free choice method of medical health care delivery within the framework of private insurance compared to a system dominated and controlled by the federal government. The Board of Trustees was granted a degree of flexibility in dealing with Congress on this issue within the perimeters of established policy.

Physician's Role in Executions

Considerable media attention was devoted to a judicial council report regarding physician participation in executions. This was a timely report since four states have laws providing executions by lethal injection of a pharmacological substance.

The House adopted the report prohibiting a physician's participation in an execution. However, a physician may continue to make a determination or certification of death as currently provided by law in any situation. The report also states that an individual physician's opinion on capital punishment is the personal, moral decision of the individual.

Kansas Reception

Although Kansas-sponsored candidates for AMA Councils (Reals and Conard) were unsuccessful in this our first bid for national office, the reception accomplished several important objectives. It was very well attended and a significant number of con-

tacts with other delegations was made at that time. The present custom of campaigning for positions on AMA boards or councils is costly. A study has been initiated to consider alternative methods for the election process.

The reception was greatly enhanced through the presence of — in addition to the official AMA Delegation from Kansas — such notable, respected, and well-known leaders as Drs. John Travis, George Burket, Donald Goering, Warren Meyer and Emerson Yoder, all accompanied by their spouses. Also present for the occasion was the leadership of the KMS Auxiliary: Mmes. John D. Huff, Robert Moore, Frank Bichlmeier, and Chester Young. All guests were greeted and received in accordance with Kansas hospitality and friendliness.

AMA House meetings provide a unique educational opportunity. *Any member of the Association* may present testimony at the reference committee hearings and, of course, corridor discussions on the issues provide ample opportunities to present diverse viewpoints. If you cannot come to the meeting, you are still represented by your delegates. Let your delegation know your opinion. You can also prepare a resolution and request that it be submitted to the House. AMA policies are often initiated by an individual physician who had a good idea and coaxed it through the democratic process.

Arrangements are customarily made during the AMA Meetings for delegates to participate in hometown radio interviews. Each interview is then distributed within 24 hours to radio stations in the delegate's hometown, and sometimes to statewide radio networks. The focus at this time was on subjects such as the impact of inflation and unemployment on the patient's ability to pay for medical care, on cost containment, preventive medicine or "wellness," trends toward primary care, and the impaired physician. There were 110 AMA members participating in the interviews while attending the 129th Annual Session, and included Drs. Scott and Purinton from your delegation.

Thank you for giving your delegates the opportunity to represent you and to present this report.

Clair C. Conard, *Delegate*
Alex C. Scott, *Delegate*
Lew W. Purinton, *Alternate*
Kermit G. Wedel, *Alternate*

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RICHARD L. SWEET, M.D., University of California at San Francisco and San Francisco General Hospital.

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JOHN M. HIEBERT, M.D., University of Virginia Medical Center, Charlottesville.
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PROFESSOR IAN McCOLL, M.S., F.R.C.S., F.A.C.S., University of London and Guy's Hospital, London, England.
MARSHALL J. ORLOFF, M.D., University of California, San Diego, School of Medicine.
RICHARD L. SIMMONS, M.D., University of Minnesota Hospitals, Minneapolis.

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At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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Tenuate® Ⓒ
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Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon (Dillon), R.H., and Leyland, H.M.: A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404

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*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

1. Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

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Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
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Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness ar sedation. Constipation, dizziness, headache, and fatigue have been reporte Generally these effects tend to diminish with continued therapy. The followi reactions have been associated with the drug, some of them rarely. (In son instances an exact causal relationship has not been established.) These includ Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities liver function tests; one report of possible drug-induced hepatitis without icter and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chl thalidone and papaverine hydrochloride. Weight gain, transient elevation of blo glucose, or serum creatine phosphokinase: congestive heart failure, Raynau phenomenon; vivid dreams or nightmares, insomnia, other behavioral change nervousness, restlessness, anxiety and mental depression. Also rash, a gioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associat with a rash, impotence, urinary retention, increased sensitivity to alcohol, drynes itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomast weakly positive Coombs' test, asymptomatic electrocardiographic abnormaliti manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or a sent reflexes and vomiting followed the accidental ingestion of Catapres (clonidi hydrochloride) by several children from 19 months to 5 years of age. Gast lavage and administration of an analeptic and vasopressor led to complete r covery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minu intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) ov dosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 m (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Al available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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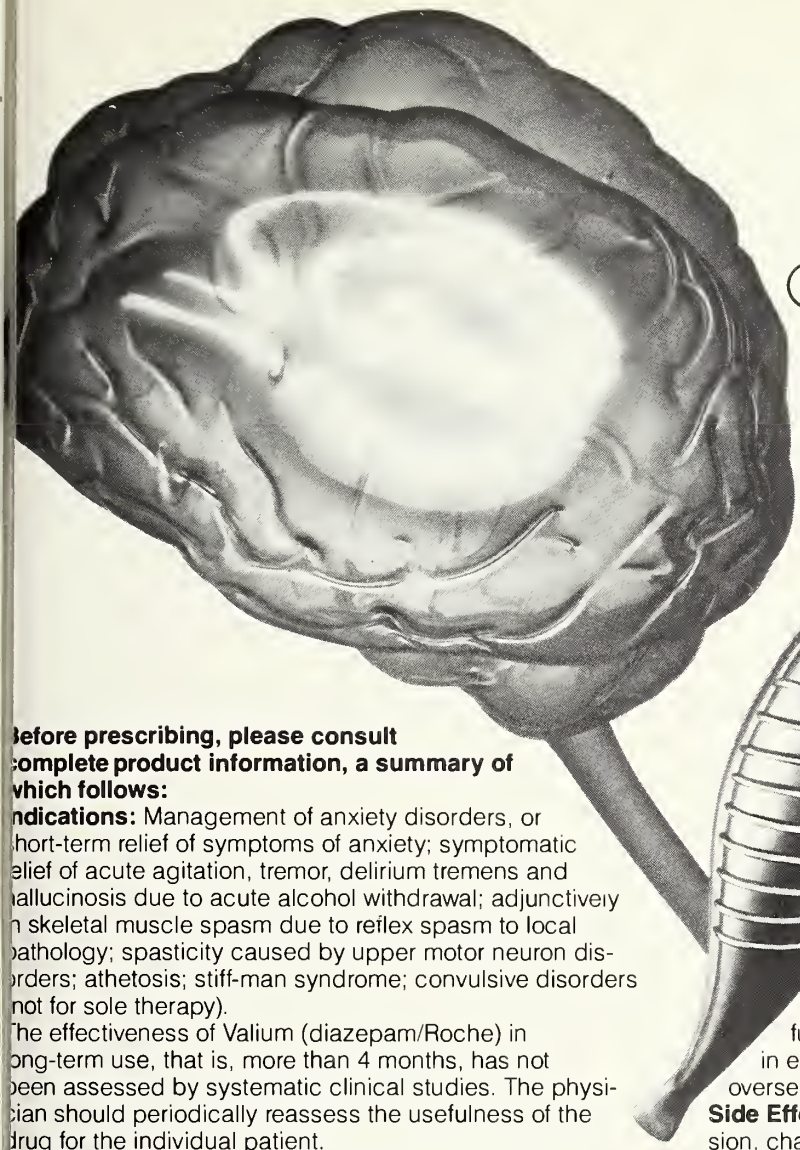
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Before prescribing, please consult complete product information, a summary of which follows:

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The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam/Roche) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available in trays of 10.

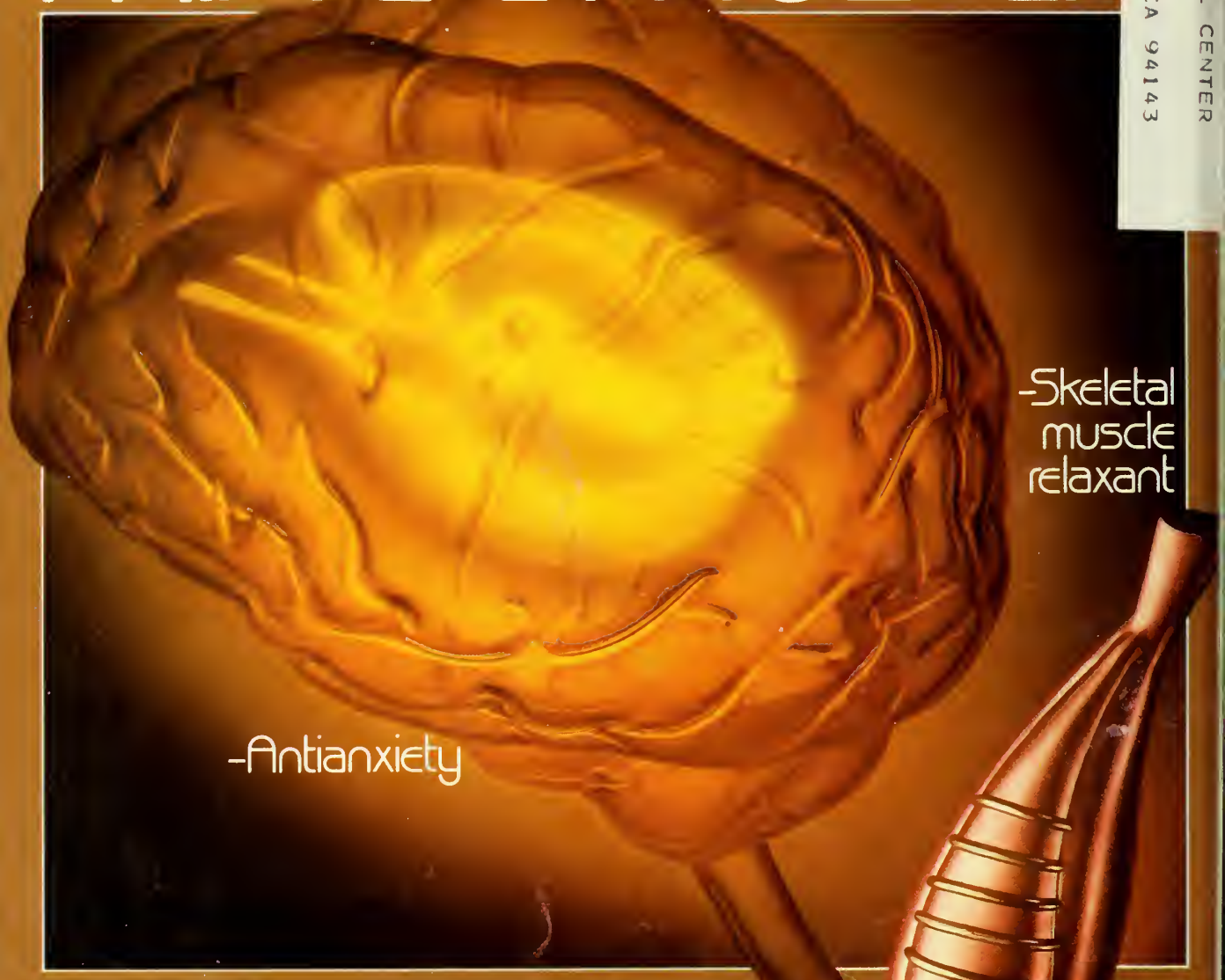
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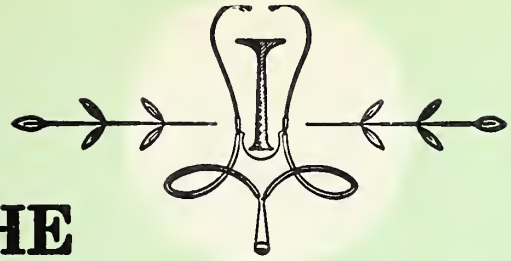
Valium®

diazepam/Roche

Indicated in anxiety disorders and as an adjunct in the relief of skeletal muscle spasm.

Please see summary of product information on preceding page.

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Medical-Legal Issue

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American Medical Association Auxiliary
535 No. Dearborn St., Chicago, IL 60610

AUXILIARY NEWS

An Open Letter to Kansas Physicians

I am grateful for this opportunity to describe activities at the University of Kansas Medical Center that are supported by AMAERF funds. It gives me a chance to inform the physicians and Auxiliary about student life at the School of Medicine as well as to express appreciation for the continuing contribution of these funds.

Enrollment at UKSM is nearing 800, with all students now in a four-year curriculum. The level of preparation of our incoming students is higher than ever, and their academic challenges remain high. An increasing number of students have roots in smaller communities across the State of Kansas, and more women are enrolled than at any time in our history, now comprising 25 per cent of the total student body. The new first-year class includes 58 women — 28 per cent of all Year 1 students.

AMAERF funds are channeled into nearly two dozen separate activities in six general categories: student awards, organizations, research days, student-faculty interactions, student recreational activities, and counseling. AMAERF provides academic scholarships to medical students who excel in their studies. Thirteen awards of $1,500 each are distributed to selected students, representing each level of undergraduate medical education, as designated by a special committee. In addition, these funds support special awards for students and faculty at commencement ceremonies.

The depth and variety of biomedical research projects here attract many medical students and strengthen their appreciation for and understanding of the scientific basis of modern medicine. AMAERF funds reinforce this vital area by supporting medical student Research Days, a highly regarded annual event that includes presentations by students and visiting scholars.

Student organizations play a significant role in student life on this campus. They serve as forums for discussion of problems; as valuable sources of feedback about the quality of instruction; and as organizers of special seminars with guest speakers. The Medical Student Assembly conducts an annual social event — The White Knight's Ball — for medical students and faculty. AMAERF funds support all of these activities. They also support travel by representatives of medical student organizations to national meetings, such as the Organization of Student Representatives of the Association of American Medical Colleges. A comparatively new medical student group supported by AMAERF funds is the American Medical Women's Association. This group plays an important role in addressing the problems faced by women in medical school.

Periodic interactions between our students and faculty in a non-academic setting have had positive, reciprocal effects in the past few years, *e.g.*, breakfast and luncheon meetings, class parties, and a graduation brunch at Spencer House. AMAERF has supported these as well as funding the annual student picnic, furnishing a new classroom, organizing an annual holiday party in December, and providing refreshments for incoming medical students during orientation week. Students at UKSM are seriously deprived of convenient and economical facilities for various recreational activities, and YMCAs and elementary and high school gymnasia are partially secured for occasional student use. With more than 2300 health care students enrolled here, the absence of recreational facilities becomes a major concern. Stopgap measures are about all that can be done

Auxiliary President, Evelyn Huff, invited David Waxman, M.D., to write this message regarding the utilization of AMAERF funds at the University of Kansas School of Medicine. Dr. Waxman is Executive Vice Chancellor of the University of Kansas College of Health Sciences and Hospital.

half-life

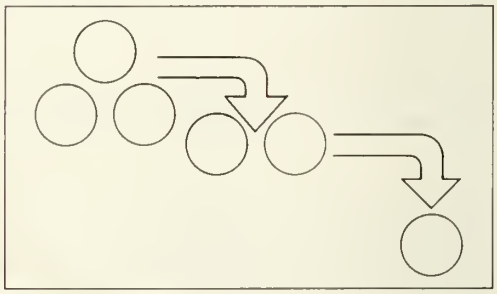
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Ensures smooth therapeutic effect even if a dose is missed The relatively longer half-life of Valium® (diazepam/Roche) has important clinical and pharmacological implications. Steady-state levels generally are reached within 5-7 days with no further accumulation. At this plateau, the patient benefits from the consistent, steady response you expect. Sharp blood level variations, frequently attributed to agents with a short half-life, do not appear with Valium.

Avoids sudden symptom breakthrough Once steady-state levels are achieved, sudden reemergence of symptoms is unlikely. Diazepam and its active metabolites exhibit overlapping half-lives that are advantageous not only during therapy but especially when pharmacologic support is discontinued. Elimination rates are gradual with Valium and thus provide a compatible adjustment interval for the patient. In comparison, blood levels of short-acting agents with inactive metabolites decrease more rapidly and are more likely to be associated with withdrawal symptoms if medication is stopped abruptly.* With Valium unwanted effects other than drowsiness or ataxia are rare. Patients should be cautioned about driving and advised to avoid alcohol.

Tapers naturally; complements gradual dosage reduction at discontinuation When any psychoactive medication is discontinued, it is good medical practice to gradually reduce the dosage. From your own experience you know this is rarely necessary after a short course of Valium therapy, but for patients on extended therapy, gradual reduction of dosage is advisable. This regimen, along with the self-tapering feature of Valium, provides a smooth transition to independent coping.

*Sellers EM: *Drug Metab Rev* 8(1):5-11, 1978



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effective therapy through efficient pharmacodynamics

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Valium®(V)
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Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome; convulsive disorders (not for sole therapy)
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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under present circumstances, and fortunately, AMAERF funds have facilitated some of these arrangements.

A unique, volunteer student counseling service has been organized at the school. An adjunct to the faculty advisory system and to student health services, the counseling group includes faculty and other staff who are experienced in counseling and who make time available to meet with students. This service has made it possible for students with personal problems to discuss them in a confidential setting with individuals who are not associated with their academic programs. AMAERF funds have contributed to the activities of this important support group.

I hope this gives you a clear picture of the scope of our efforts involving AMAERF funds. While we always hope for more, needless to say, we are deeply grateful for what we receive. Many, many thanks.

Sincerely,
David Waxman, M.D.
Executive Vice Chancellor

PHYSICIAN PLACEMENT DIRECTORY

The American Medical Association's Physicians' Placement Service has released its August, 1980, edition of the *Opportunity Placement Register* which lists thousands of openings for physicians and is indexed by specialty, type of practice, location and size of community, date of availability, financial arrangements, and other data.

For those communities seeking the services of a physician, the August, 1980, edition of the *Physician Placement Register* is also available. It contains the coded résumés of physicians who wish to relocate.

If a physician is looking for a new practice location — or if a community is seeking the services of a physician — the AMA's Physicians' Placement Service is a source of information, providing a broad view of the marketplace.

For more information, contact the Physicians' Placement Service, AMA Headquarters, 535 N. Dearborn St., Chicago, IL 60610.

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(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.
Final classification of the less-than-effective indications requires further investigation.
The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticanal or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

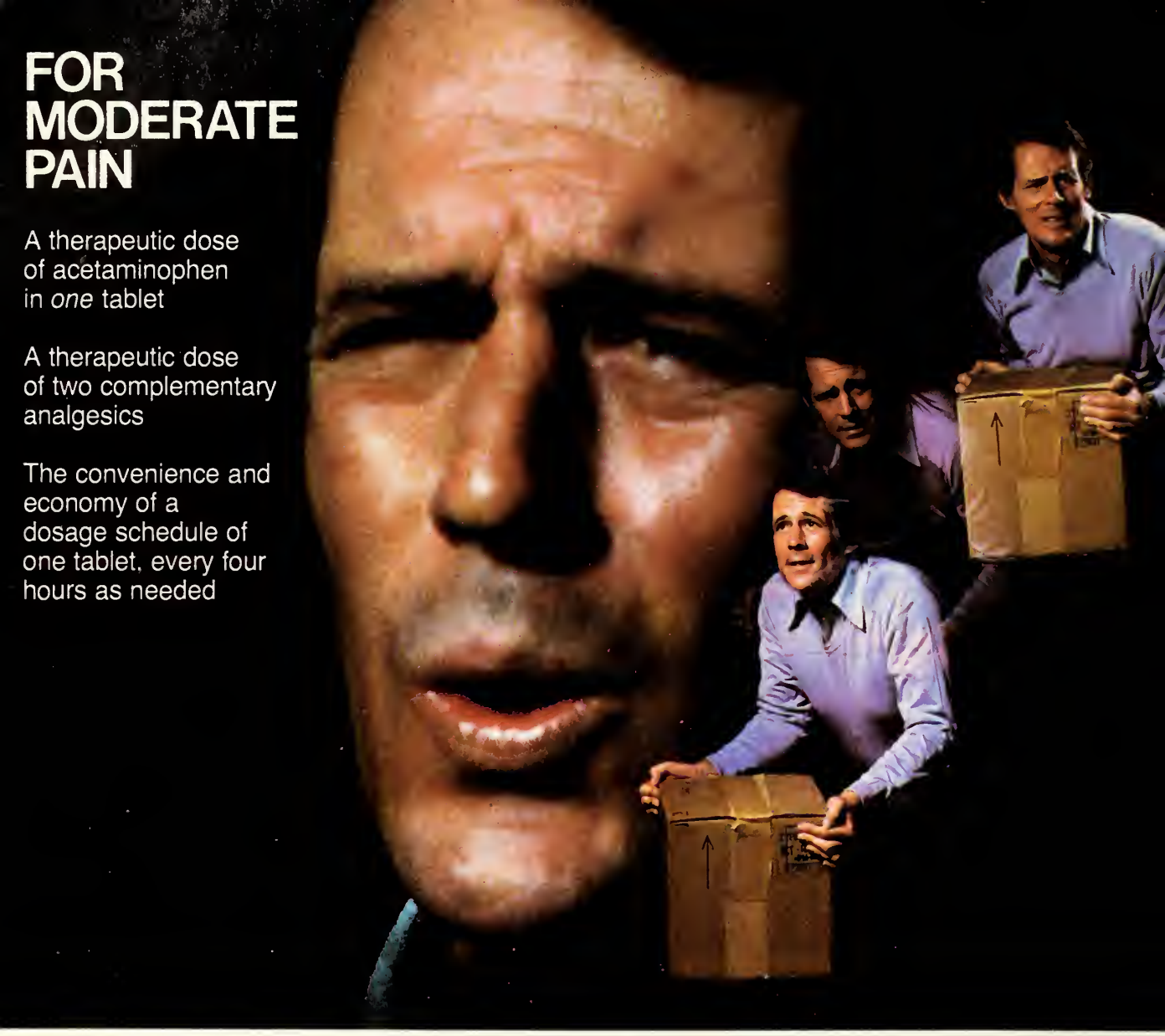
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WYGESIC—Abbrevieted Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and or physical abilities required for potentially hazardous tasks, e g driving a car or operating machinery Patients should be cautioned accordingly

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions Cases of liver dysfunction have been reported

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. (see **Warnings**) Confusion, anxiety and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours However, early recognition may be difficult since early symptoms may be mild and nonspecific Evidence of liver damage is usually delayed After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered, preferably IV, simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory In addition to a narcotic antagonist, the patient may require careful titration with an enticonvulsant to control seizures Analeptic drugs (e g caffeine or amphetamine) should not be used beceuse of their tendency to precipitate convulsions

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated Gastric lavage may be helpful Activated charcoal can absorb a significant amount of ingested propoxyphene Dialysis is of little value in poisoning by propoxyphene alone Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed Copious gastric lavage and or induction of emesis may be indicated Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness Appropriate literature should be consulted for further information (JAMA 237 2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity Serial hepatic enzyme determinations are also recommended

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Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories — Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream — Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories — boxes of 12 (N 0047-0089-12) and boxes of 24 (N 0047-0089-24) in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream — one-ounce tube (N 0047-0090-01) with plastic applicator.

Store between 59°-86° F (15°-30° C).

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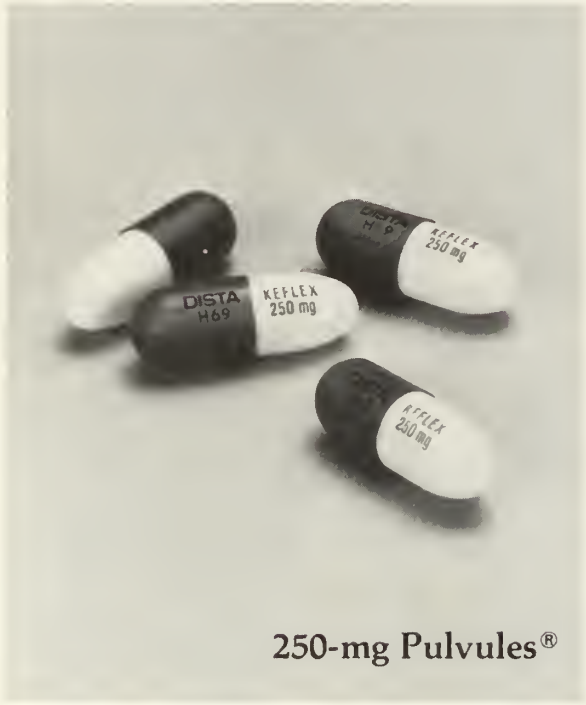
The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

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Chlorpheniramine Maleate	8 mg
Hyoscyamine Sulfate	0 19 mg
Atropine Sulfate	0 04 mg
Scopolamine Hydrobromide	0 01 mg

Ru-Tuss Tablets act continuously for 10 to 12 hours.
Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation

INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets

CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated

WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers

PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings.)

OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure

DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole

HOW SUPPLIED

Bottles of 100 Tablets	NDC 0524-0058-01
Bottles of 500 Tablets	NDC 0524-0058-05

Federal law prohibits dispensing without prescription

COUGH

RU-TUSS® EXPECTORANT

DESCRIPTION
Each fluid ounce of Ru-Tuss Expectorant contains

Codeine Phosphate	65 8 mg
(WARNING. MAY BE HABIT FORMING)	
Phenylephrine Hydrochloride	30 mg
Phenylpropanolamine Hydrochloride	20 mg
Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
Alcohol	5%

Ru-Tuss Expectorant is an oral antitussive, antihistaminic, nasal decongestant and expectorant preparation

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold

CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an antihypertensive or antidepressant drug containing a monoamine oxidase inhibitor is contraindicated.
Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthma and in women who are pregnant

WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patient should be warned of the potential that this drug may be habit forming. Ru-Tuss Expectorant may cause drowsiness. Patients should be warned of the possible additive effect caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers

PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease. Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions

DOSAGE AND ADMINISTRATION Adults 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.
Children 6 to 12 years of age ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age ½ teaspoonful every 4 hours, not to exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age. Use as directed by a physician

HOW SUPPLIED

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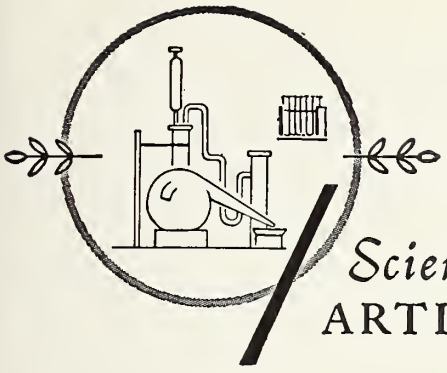
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Regulation and Control

The Function of Law in Medicine

HOWARD N. WARD, M.D., J.D.,* ***Topeka***

How and why does society and its government subject its physicians to regulation and control? Dealing with such a broad topic in a brief essay is at least difficult and perhaps foolhardy. Nevertheless, even a rudimentary understanding seems worth while. An admittedly incomplete definition of the law offered by Waltz and Inbau is "the body of authorized rules by which a civilized society regulates, minimally, the conduct of its members."[1] The purpose of the law is to keep society-at-large, and special segments of it, within reasonable behavioral bounds in order to prevent society from flying apart and becoming chaotic. The physician, first of all, is subject to those laws and regulations that apply to every member of society and no attempt will be made to outline this vast body of material. Secondly, the physician is subject to those statutes, rules, regulations, and decisions that deal with medical practice. This body of law is collectively termed medical jurisprudence, and represents the impact of law and legal process on the practice of medicine. It is distinguished from forensic medicine, which deals with the impact of medical principles on the practice of law. The medical profession has been intimately entrusted with the lives and well-being of society's members, and consequently society-at-large has a tremendous stake in its behavior and performance. The people — the source of power in our representative form of democracy — have by their governments laid down rules for those who wish to engage in this particular occupation. Hence, mandatory licensure and compliance with the law is the price that must be paid for the privilege of practicing medicine. To argue otherwise would be to assert that the medical profession is above or outside of the scope of the law and the sovereignty of the people. Of course, the people — by their government — could have delegated to organized medicine the responsibility and power to regulate the profession, but they have not done so. The purpose of the Kansas licensure requirement is given in Kansas Statutes Annotated 65-2801 which states as follows:

Law functions to keep society within reasonable behavioral bounds. Discussed here is that portion of the body of law — medical jurisprudence — that represents the impact of law and legal process on the practice of medicine.

> Recognizing that the practice of the healing arts is a privilege granted by legislative authority and is not a natural right of individuals, it is deemed necessary as a matter of policy in the interests of public health, safety,

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and welfare to provide laws and provisions covering the granting of that privilege and its subsequent use, control, and regulation to the end that the public shall be properly protected against unprofessional, improper, unauthorized and unqualified practice of the healing arts, and from unprofessional conduct by persons licensed to practice under this act.

Physicians are often irritated, and occasionally incensed by the intrusion of law into medicine, perhaps partly because he or she does not understand its purpose. The difference between the knowledge and skills of a physician as compared to a layman with respect to medical matters is so great that it would be possible for the unscrupulous, unprofessional, and even the ill-qualified within the profession to overreach and take unfair advantage of society were there no regulatory and disciplinary provisions. In other words, as stated by Foote, the physician with his unique training, responsibility and power over life itself, is subject to certain expectations, privileges, and controls in society that do not apply to others.[2] Thus, notwithstanding the assertion in the Declaration of Independence* that we are all endowed with certain unalienable rights which include life, liberty, and the pursuit of happiness, these rights cannot be asserted to the extent of permitting one to deliberately or negligently harm others.

Another fundamental role that law plays in our society is conflict resolution by negotiation and settlement as well as litigation. It is a way of substituting a civilized process for the sword and cannon. This is not to say that most conflicts between persons or persons and institutions cannot be resolved by non-judicial methods. Hopefully, the majority of conflicts could be resolved short of legal consultation or litigation. However, our society does appear to be litigious at present and this modern trend toward "lawsuit mania" has been aptly named "Litigatus Americanus" by the then Judge and now Professor Michael Barbara.[3]

Medical Responsibilities

At the risk of oversimplication, Foote divides specific medical responsibilities to the law into three categories. The first — physician-patient responsibility — concerns the art and science of healing. An example would be the selection of a proper antibiotic for treatment of pneumonia. Here, the law, within certain boundaries, will not second guess the physician. The boundaries are determined by the standard of care usually established by medical peers in the role of expert witnesses. The second category deals with physician-society problems. An example would be the maintenance of sanitary conditions in the hospital or office. The physician has no particular monopoly on that type of knowledge, and the law is not reticent to require specific standards. Hence, the boundaries allowed a physician's judgment are more restrictive. The third category is most complicated since it deals with physician-patient-society responsibilities. Concerns in this area include such issues as abortion, euthanasia, and human investigation to mention only a few. Here, serious ethical, philosophical, financial, and religious questions come into play that transcend a mere diagnostic or therapeutic decision made principally — and often exclusively — for the benefit of a single patient. Again, the law and society do not subscribe to the belief that the medical profession has cornered all of the world's wisdom on such difficult questions. Consequently, the physician is again more strictly regulated than in the first category.

Reimbursement

A more recent intrusion of law upon medicine involves the regulation of reimbursement rather than the practice of medicine. By determining the time, method, and even extent of payment for services rendered to patients covered by state and federal programs, physicians are being controlled for motives that seem different from those previously mentioned. Although these programs involve quality control procedures, one senses that other motives are given a higher priority. These other goals appear directed toward cost containment by structuring the quantity of care that can be delivered and the level of payment that can be dispersed for a given service. It would seem that the physician of the future will have to be an astute negotiator of contracts in order to make certain that rewards are commensurate with level of training and responsibility in society. Although the goals of cost containment do not seem as laudable as those that concern the quality of the practice of medicine, they are equally real in this era of economic recession coupled with the recognition that we are a nation of limited resources. Also, even though some may question the right of the government to regulate medicine through the "backdoor," there appears to be no question but that it has the power. Hence, reimbursement methods and amounts and cost containment programs and procedures will, no doubt, require an ever increasing amount of physician time and effort if physicians are to receive equitable treatment with respect to the nation's resources.

* Although this document has no legally binding effect, its spirit and principles are sometimes invoked in interpreting the Federal Constitution.

Interprofessional Relations

It is becoming apparent that psychiatry and the law are entering an adversary status because of the conflict between patient's needs and patient's rights. Furthermore, another cascade of medical malpractice suits will almost certainly exacerbate the level of distrust and hostility between the two professions. This appears to be true, even if the majority of suits are meritorious since being a defendant is a very unpleasant experience. The personal agony, social embarassment, and loss of time and income associated with litigation are not easily dismissed as being mere side effects of the American way of solving problems. This is especially true if the physician believes the claim to be unjustified. Furthermore, if a physician believes he must view each patient as a potential adversary, then this has an unfavorable effect on the physician-patient relationship by eroding the trust usually placed in a fiduciary and provoking the defensive practice of medicine with its attendant excessive testing, over-hospitalization, and an inclination to avoid high risk procedures. Contrariwise, to blindly march ahead in care of patients discounting any fear of litigation can lead to inadvertent gaps in medical records that invite liability. Because of this, some believe that applying the adversary process to medicine is an unworkable or "wrong key" approach. Mellinkoff suggests that remedies more rational than the adversary system could be evolved to safeguard patients and ease their woes.[4] He further argues that the adversary method must stop short of malpractice insurance premiums that would make old King Midas sigh or cause Hippocrates to become a full time farmer. Unfortunately, he offers no alternative except to quote Abba Eban who stated "both men and nations do behave wisely once they have exhausted all other alternatives." An option often mentioned is a no-fault approach to medically related injuries analogous to worker's compensation for injuries that arise out of the employer-employee relationship. However, according to Mills, the cost of compensating patients for all medically related injuries, whether caused by negligence or not, would be several times that of costs incurred through the adversary system if the awards were comparable for a given injury.[5] In addition — as witnessed in worker's compensation cases — the elimination of fault as a criterion for an award does not eliminate argument and dispute since the employer may argue that the injury did not arise out of, and in the course of, the employer-employee relationship. Similarly, patients might allege injuries arose during the course of medical care when in actuality, the injuries occurred before or after the rendition of medical care and the contest would be in full bloom. Therefore, the adversary system may be all that society can afford, and many attorneys would also argue that it is still the best method that man has devised for flushing truth into the open. Hence, at present, the best option for physicians is as well stated by Pollack as by anyone: "The golden mean between fear and overconfidence is to know where the risks lie and to take reasonable precautions against them."[6] Many of the guidelines for avoiding liability published nearly 20 years ago still have substantial validity.[7]

The term medical malpractice rings too harsh when one looks at the nature of many of the claims. The prefix "mal" is defined in Black's Law Dictionary as bad, wrong, or fraudulent.[8] It appears that the meanings of "bad" and "fraudulent" imply an unseemly departure from the practice of medicine which, in actuality, is often not all that bad and rarely fraudulent. The term "wrong" is acceptable and generally more appropriate, but unfortunately is not the meaning engraved on the minds of many people. Perhaps those cases involving an injury arising from medical care could be labeled *medically related injury* analogous to the term "personal injury" which is used in many accident cases. The point is, that the very term "medical malpractice" is inherently inflammatory.

Another reason for hostility between the two professions could be a lack of understanding of the methods and modes of thinking of both walks of life. Actually, there are many similarities as well as differences. Both professions require extensive and continuing education, both demand ideally the highest kind of integrity, both deal with complex, difficult and often insoluble human problems, both are capable of challenging the best intellects in an academic sense, both offer great satisfactions in the practical sense, and both have noble and rotten-apple members. To be humorous, someone once said that lawyers are just like doctors: what one says, another contradicts. However, there are also substantial differences. The most significant is that although the physician serves his/her patient and the attorney serves his/her client, the attorney must also deal with the conflict between the person served and one or more other parties.[2] In the case of the physician-patient relationship, the culprit is disease, and nobody likes disease or injury. Everyone — especially the patient — cheers the physician in this fight. There is no dissent. The attorney becomes involved in the client's problem only in the context of third parties and always under an umbrella of conflict; that

is, his client has an adversary — another person or institution — and as an advocate of his/her client, the attorney is obligated to present the case to the best possible advantage of the client within the bounds of the law. As Foote points out, if one strips away the veneer, the difference between an attorney in the court room and a medieval soldier with a lance representing his "client" is one of tools only. Hence, a trial can be a competition of two or more inconsistent versions of the facts and differing theories of law as well as a race for credibility. Another significant difference is the nature of the diagnostic process versus judicial decision making.[9] The physician is looking to the immediate good of a single patient (except in obstetrics), whereas the legal system must reach a decision that comports not only with the parties involved, but with society-at-large. The law's rightful concern with the larger community sometimes causes the result in a given individual factual situation to be harsh. The physician uses the scientific method to reach the one best diagnosis for the patient's dilemma. The attorney seeks to apply settled principles of law (statutes and precedent established by previous decisions) in the most favorable way to his/her client's factual situation, or if the case so requires, to argue that a statute is for some reason invalid or that precedent should be overturned. Because an attorney cannot always anticipate on which side of a contested issue his next client will be, he is carefully trained to recognize the favorable and unfavorable aspects of both sides of given factual situations. This also is of great importance in trying a case since it permits one to know the strengths and weaknesses of the other side. Thinking like a lawyer requires one to understand both sides of the case. Contrariwise, the physician is trained to progressively narrow the range of possible diagnoses to the one best answer. The process of differential diagnosis is perhaps slightly akin to thinking like a lawyer since it involves weighing the evidence for and against a given number of possible answers to the patient's malady. However, the attorney is not privileged to know the final answer until the verdict is reached by the trier of facts. The law is usually concerned with redressing past wrongs with all of the facts fixed. The physician is dealing with current biological events which are dynamic and changing, thus requiring him to make a prediction (prognosis) regarding future events with and without therapeutic intervention. The law deals with human nature and man's intent, and medicine deals with nature itself and the ability to intervene and alter the natural course of disease. Although medicine is an imperfect science as compared to physics, it is more certain and predictable than is the outcome of legal procedures, such as adjudication of contested matters.

In 1958, an Interprofessional Code for Law and Medicine (see appendix) was jointly adopted by the Kansas Bar Association and the Kansas Medical Society. The code is based on the principle that a substantial number of persons need the services both of a physician and an attorney, and that the welfare of the physician's patient and the attorney's client are best served when there is cooperation between the two professions. It provides for common courtesies and sets guidelines for reports, medical testimony, and compensation.

Appendix

Kansas Interprofessional Code for Attorneys and Physicians

(The Interprofessional Code for Attorneys and Physicians was enacted by adoption by the Kansas Bar Association and the Kansas Medical Society in 1958. It was published in Vol. XXVII, JKBA 62 (August 1958) and in the November 1978 Kansas Barletter. The Code is reprinted courtesy of the Professional Relations Committee of the Kansas Bar Association. Additional copies may be obtained by contacting the Kansas Bar Association office, Box 1037, Topeka, Kansas 66601.

Interprofessional Code for Attorneys and Physicians

A. Preamble

Acknowledging that a substantial part of the practice of law and medicine is concerned with the problems of persons who are in need of the combined services of a lawyer and doctor; that the public interest and individual problems in these circumstances are best served only as a result of cooperative efforts of all concerned; that members of both the legal and medical professions share an obligation to the individual and to society, we, the members of the Bar Association of the State of Kansas and the Kansas Medical Society, do adopt and recommend the following declaration of principles as standards of conduct for attorneys and physicians, in interrelated practice.

B. Cooperation Between Professions

1. It is recognized that the welfare of the physician's patient and the attorney's client requires complete cooperation between the physician and the attorney in all cases involving their combined services.

2. A physician should not advise on the amount of damages a patient should seek to recover, nor upon

whether or not to employ an attorney. An attorney should not ask a physician to form or express an estimate of money damages in behalf of a patient.

3. It is recognized that an attorney is the advocate of his client, and does not and cannot properly represent both sides. It is also recognized that a patient's attending physician has an obligation to furnish the patient's attorney with proper medical facts.

C. Reports and Conferences

1. There should always be one or more conferences between the physician and attorney relative to the common problems in a particular case. Arrangements for conferences should conserve the time of all parties and should be held at the mutual convenience of the physician and attorney.

2. The physician should feel obligated to point out anything which he believes will be helpful in presenting the patient's case and any weaknesses in opposing medical theories or testimony.

3. It is the obligation of the attorney to arrange desirable conferences and to apprise the physician of the significance to the case of the particular medical testimony involved. The attorney should always invite and answer all questions of the physician concerning the evidence or its presentation, and should counsel the physician relative to cross examination.

4. A physician is entitled to the written authorization of the patient before he furnishes any attorney information concerning the history, physical condition, diagnosis or prognosis of a patient, and the attorney should furnish such written authorization in advance of a request for a conference or medical report.

5. The patient's attorney is entitled to a prompt report from the attending or treating physician concerning the medical facts, including history, treatment, diagnosis and prognosis. It is the obligation of the attorney to outline to the physician the matters he deems desirable to be covered in a medical report. A medical report shall be furnished as promptly as possible.

D. Medical Testimony

1. The physician recognizes that medical testimony is often absolutely essential.

2. The attorney should make arrangements to permit the doctor to testify at the most convenient time and without unavoidable delay in the courtroom.

3. It is recommended that a conference should always be held prior to testimony.

4. If an attorney plans to have a subpoena served on a physician he should notify him, preferably in advance, of service where circumstances permit. The physician recognizes that in many cases justice requires the service of a subpoena.

5. It is recognized that the administration of justice by the courts cannot depend upon the convenience of litigants, attorneys or witnesses, including physicians called to testify. Therefore:

(a) The attorney should notify the physician as far in advance as possible as to when he is to be needed to testify, and keep him notified and advised as to any changes in this respect as they arise.

(b) The physician should arrange to appear promptly when requested and do so unless prevented by circumstances which would constitute legal excuse.

6. The physician, while testifying should:

(a) At all times maintain the dignity of his profession.

(b) Answer questions as concisely and objectively as possible, using terminology, when permissible, which is understandable to a jury of laymen.

(c) If he does not know the answer to any question, so state and make no attempt to conjecture or theorize, or give answers not responsive to questions propounded or volunteer testimony.

(d) Under no circumstances permit any bias, prejudice, favoritism or personal interest to influence his testimony.

7. The attorney, in examining or cross-examining a physician, should:

(a) Avoid questions which browbeat or badger the physician. The physician recognizes that it is the attorney's obligation to his client to be responsible for the trial of the case, and recognizes that cross examination is a necessary part of the process of justice.

(b) Prepare and propound all questions to the witness in such form and manner as will permit clear understanding and a forthright answer.

(c) Cooperate with the physician by minimizing, as far as practicable, the time required for the physician to remain in court.

8. It is recognized that there are two categories of medical testimony:

(a) A doctor testifying for *his patient*.

(b) A doctor testifying as an *expert witness*, ordinarily as to party who has not been his patient.

In the former case the physician has an obligation to his patient to cooperate with the patient's attorney

in presenting the medical facts in court. A physician, however, asked to testify as an expert witness not involving his patient is under no obligation to do so unless he so desires.

E. Compensation for Services of Physicians

1. A physician is entitled to reasonable compensation for professional services rendered. An attorney is prohibited by the ethics of his profession from making payment to the physician for medical reports or testimony. Such compensation must be paid by the patient or client. The physician is prohibited by the ethics of his profession from entering into any arrangement whereby the physician's charge is determined by the amount of financial recovery.

2. Notwithstanding that the attorney cannot assume an obligation to pay the fees of a physician, it shall be the obligation of the attorney to ask authority of the patient to pay the physician direct for his services out of any money recovery obtained for the patient. It shall also be the obligation of the attorney to cooperate with the physician in making all proper arrangements for payment for his services.

3. When a physician testifies as an *expert witness* as distinguished from testifying in behalf of his patient, his fees will be paid by the party calling him, in such amount as shall be agreed upon with the attorney representing such party.

4. Although the physician must set his own fees, it is suggested that a reasonable standard in many cases would be the equivalent of what the charge would be to a patient for the same amount of time and skill for professional services.

5. No attorney shall charge a fee to a physician for collection of an account for medical services, collected in personal injury litigation for the client-patient.

F. Joint Medical-Legal Committee

The Bar Association of the State of Kansas and the Kansas Medical Society shall each appoint six members from its profession who shall serve on a committee, two to serve for a term of one year, two for a term of two years, and two for a term of three years and thereafter members shall be appointed a term of three years, and said twelve individuals shall constitute the Joint Medical-Legal Committee. Such committee shall:

(a) Promulgate such suggestions as may be necessary to carry into effect the principles hereby adopted.

(b) Jointly attempt to mediate and arbitrate, in the first instance, any disputes arising between individual physicians and lawyers or between the two professions.

(c) Report annually to each of said organizations the work of the committee during the year and make such recommendations to said organizations as the committee deems desirable.

G. Enactment

This code shall become effective upon its adoption by the Bar Association of the State of Kansas and the Kansas Medical Society. It shall be subject to amendment by joint action of the two professions and shall guide both professions in their interprofessional relationships in a spirit of cooperation and understanding.

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Medical Malpractice

The Kansas Law

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A MALPRACTICE action is a claim by a patient that a professional service was improperly or negligently rendered. As distinguished from licensing statutes designed to regulate the practice of medicine or criminal statutes which punish for statutory violations, the law of medical malpractice exists to provide a mechanism to compensate a patient for an injury sustained by the improper or careless practice of the profession. Fundamentally, such actions are identical, whether the profession involved be medicine, architecture, engineering, law, or any other specially recognized and licensed profession. What follows is an explanation of the Kansas Law of Malpractice.

It is interesting to observe that the development of the law of malpractice has followed the evolution of society. Medical treatment has been the subject of regulation almost from the beginning of organized government.

The oldest known code of law — the Code of Hammurabi dated 2200 B.C. — in effect, provided for the organization and control of the medical profession. Its contents included what we now call medical malpractice, and it proposed, for the first time, civil and criminal liability for medical personnel. Penalties ranged from monetary compensation to cutting off the surgeon's hand.[1]

The Visigothic Code of the fifth to seventh centuries imposed severe restrictions on medical personnel, similar to the Code of Hammurabi. Before undertaking a case, the physician was required to enter into a contract and give security; if death ensued, he received no fee. An injury to a nobleman during venesection brought a penalty of 100 solidi. If the nobleman died, the physician was turned over to the relatives who might deal with him as they saw fit. If the medical man killed or injured a slave, he was required to replace the slave with another of equal value.[2]

The Justinian Code, 529-564 A.D., regulated the practice of medicine and surgery. The code called for, among other things, an examination to test the physician's professional competency, a limitation on the number of physicians, and penalties for malpractice.[3]

One of the earliest recorded malpractice actions in England was in the late 14th or early 15th century when a petition to the Chancellor requested him to appoint "the surgeons of our Lord the King, and by other sufficient surgeons . . . " to examine the petitioner to establish the truth of his claim that a surgeon set his arm improperly.[4]

The development of the law of malpractice has followed the evolution of society. Medical treatment has been the subject of regulation almost from the beginning of organized government. This discussion relates to the Kansas Law.

The first reported American malpractice case, in 1794, was an action for an improperly performed mastectomy in which the plaintiff received a judgment of 40 pounds.[5]

Tefft v. Wilcox

The development of the law of medical malpractice, essentially as we now know it, coincided with the first such action reaching the Kansas Supreme Court. *Tefft v. Wilcox*[6] was an action brought against a Topeka physician whom the plaintiff claimed failed to properly set the plaintiff's shoulder. Tried to a jury in 1866, it resulted in a verdict of $2,900.

The Kansas Supreme Court reversed the lower court and ordered a new trial, chiefly because the trial court improperly instructed the jury upon the standard to which a physician is held.

Citing several decisions from the State of Maine and other New England states, the Kansas Supreme Court set forth principles of law which have controlled the trial of such cases, virtually unchanged, for over 110 years. Summarized, these principles are:

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1. A physician is never considered as warranting a cure, unless he enters into a special contract for that purpose. Rather, his contract, as implied in law, is that he possesses that reasonable degree of learning, skill, and experience which is ordinarily possessed by others of his profession and that he will use reasonable and ordinary care and diligence in the treatment of the case that he undertakes. Furthermore, the physician will use his best judgment in all cases of doubt as to the proper course of treatment. He is not responsible for damages for want of success, unless it is shown to result from want of ordinary skill and learning. He is not required to possess extraordinary knowledge or to exercise extraordinary diligence and care.
2. The physician may not be responsible in damages for errors in judgment or mere mistakes in matters of reasonable doubt or uncertainty. A physician's best judgment is constantly appealed to, and upon such judgment, he must rely. It is plain that there is great room for difference of opinion in the exercise of the arts of surgery and medicine, and as a result, there are usually more ways than one of accomplishing the same thing. If the physician exercises such judgment in an enlightened and reasonable manner, he will not be responsible for errors.
3. The degree of learning and skill that the physician holds himself out to possess is that degree that is ordinarily possessed by the profession as it exists at the time, and not as it existed at some time in the past.
4. The standard of legal obligation must be given to the jury and this evidence must, from its very nature, come from experts,

 > as other witnesses are not competent to give it, nor are juries supposed to be conversant with what is peculiar to the science and practice of the professions of medicine and surgery to that degree which will enable them to dispense with all explanations.

 Expert medical testimony is necessary.
5. The standard of legal obligation that the physician is held to is that settled by the authorities within his reach. The opportunities by reason of locality, or other circumstances, may be more favorable for one than another, in which event the responsibilities may differ. This first statement of what later became known as the locality rule included a quotation from *Elwell on Malpractice* to the effect that in the smaller towns and country, those who practice medicine and surgery, though often possessing a thorough theoretical knowledge of the highest elements of the profession, do not enjoy so great opportunities of daily observation and practical operations, as those who have resided in the metropolitan towns. The court stated that, although he may be just as well informed in the elements and literature of the profession, a rural physician should not be expected to exercise that high degree of skill and practical knowledge possessed by those having greater facilities for performing and witnessing operations, and who are, or may be, constantly observing the various accidents and forms of disease. The law will not, therefore, as a general thing, require so high a degree of knowledge to bring this class of physicians up to the rule or ordinary knowledge and skill as in places where greater facilities are afforded by which higher professional knowledge is obtainable.

Many of the subsequent cases in the Kansas Appellate Courts merely applied the law settled in *Tefft*. Some modifications of the doctrines may be noted in the case law, as set forth below.

The Locality Rule

As subsequently expressed, a physician is expected to possess and exercise that reasonable degree of learning and skill ordinarily possessed by members of his profession and of his school of medicine in the community wherein he practices, or in similar communities.[7] In *Avey v. St. Francis School of Medicine* the court held that the physician who did not practice in the community could become aware of the resources and standards existing in the locality and be allowed to express an opinion concerning whether a health care provider deviated from that standard of care.

In a recent decision, *Chandler v. Neosho Memorial Hospital,* [8] the court held that

> "locality" has to do with population, location, hospital and laboratory facilities, staff, and the medical practitioners and specialists available. It is simply one of the factors to be considered. A small hospital would not ordinarily have or be expected to provide specialized equipment and facilities necessary for the treatment of diseases or conditions which are rarely or infrequently found; on the other hand, it would be expected to have that basic equipment essential for the care and treatment of those diseases and conditions regularly encountered among its patients.

Advances in travel, communication and publications over the past century have markedly reduced the gap between urban and rural practitioners in all professions. Medical and other professional journals are widely and promptly distributed. Seminars, attended by practicing members of the professions from communities of all sizes, are not only commonplace but are regarded by most pro-

fessionals as essential to the continuing learning process required.

No doubt many areas of medical practice and hospital care are now standard throughout the United States. And yet other areas of medical practice and hospital care, procedures and treatment may differ because of local preference and experience, because of altitude and climate, or because of the availability or lack of equipment, facilities and staff. We are not, therefore, prepared to say that national standards apply in every area of health care; but we recognize that some standards may well be of universal application.

Thus, while the statement of law that a physician will be held to the standard of the community or similar communities continues, the decisions indicate a willingness to permit testimony of a non-resident physician if he establishes a knowledge of the applicable standard. Whether this may ever be an issue of law for the court, as opposed to one of fact for the witnesses to testify to, is not resolved by *Chandler*.

Necessity for Expert Testimony

As stated in *Tefft,* and refined by the subsequent decisions: Expert medical testimony ordinarily is required to establish negligence or lack of skill on the part of a physician unless the results are so patently bad as to be manifest to lay observers or as to come within the general knowledge and experience of mankind.[9] Examples of situations where it might be said that results of treatment were so unfavorable as to warrant an inference of negligence from lay testimony alone, when considered in the light of common knowledge and experience, are cases in which the physician failed to set a broken arm and discharged his patient before the bone had set and allowed the ends to overlap;[10] failing to remove a metal airway or tube from the throat of a patient,[11] and situations where surgical sponges were left in the patient following surgery.[12]

Another circumstance by which the requirement for expert medical testimony may be avoided is one in which the doctrine of *res ipsa loquitur* may be applied. A Latin phrase meaning "the act speaks for itself," it is a doctrine that exempts the plaintiff from producing direct evidence of negligence, and permits the plaintiff to present his case to the jury based upon the inference of negligence. It has been sparingly and restrictively applied in medical malpractice cases in Kansas.

In two cases where the rule was applied, the court emphasized that it is only in those cases where common knowledge, observation, and experience have shown that injury would not have occurred to the patient if reasonable medical or surgical care had been used, that negligence can be inferred from the injury itself, and the doctrine of *res ipsa loquitur* was thus applied.

In *Voss v. Bridwell,*[13] an endotracheal tube was inserted into the esophagus of the patient and was left there during the entire operation. As a result the patient was deprived of air and rendered decerebrate. In *Emrie v. Tice,* [14] a patient suffered severe x-ray burns to his face, head, and neck during radiation therapy for removal of a wart from his right ear.

Consent to Medical Treatment

A surgical procedure performed without consent constitutes an assault and battery for which an action may be maintained even though the surgeon was learned and skillful and exercised due care and skill in all he did.[15]

Kansas laws recognize that under emergency conditions requiring immediate surgery, when it is impossible or impractical to obtain consent because delay would endanger the patient's life or health, a physician is excused from obtaining consent and may proceed if what he does is in accordance with ordinary practice among the members of his profession in the same or similar communities under like circumstances.

Moreover, Kansas law recognizes that if during the course of a surgical operation to which the patient has consented, unforeseen conditions are discovered that necessitate further or different treatment to preserve or protect the health or life of the patient, and it is impossible or impractical to obtain the consent of the patient or someone authorized to consent for him, the physician may proceed if what he does is in accordance with ordinary practice among the members of the medical profession in the same or similar communities under like conditions.

Kansas law recognizes that there may be a substituted consent by one other than the patient himself if the patient is unable by reason of incapacity to effectively consent to the necessary medical care. In such instances consent by someone authorized to give the consent on behalf of the patient, such as a guardian or next of kin, may be accepted.[16]

In 1960, the Kansas Supreme Court decided *Natanson v. Kline,*[17] a case in which the plaintiff claimed injuries from radiation therapy. The claim asserted was that the defendant violated his duty to the plaintiff by making no disclosure of significant facts within his knowledge which were necessary to form the basis of an intelligent consent by the patient to proposed cobalt radiation treatment.

Since *Natanson,* the Supreme Court has decided at least ten cases involving questions of informed consent. As this doctrine has evolved, it is now estab-

lished that in the absence of an emergency, a physician or surgeon has a legal obligation to make a reasonable disclosure to his patient of the nature and probable consequences of the suggested or recommended treatment, and to make a reasonable disclosure of the dangers, within his knowledge, that are incident or possible to the treatment he proposes to administer, in order that that patient will have a basis upon which to make an intelligent and informed decision regarding the proposed treatment.[18] The duty of the physician is limited to those disclosures that a reasonable medical practitioner would make under the same or similar circumstances. What is a reasonable disclosure upon which informed consent may rest depends upon the facts and circumstances in each case.[19]

Where a disclosure of possible results of medical or surgical procedures has been made to a patient and is ascertainable, expert medical testimony is ordinarily necessary to establish that it was insufficient to accord with disclosures made by reasonable practitioners under the same or like circumstances. A consent form executed by the patient stating that the patient has been informed of the risks and hazards of the proposed procedure is ordinarily sufficient to cast the burden upon the patient to produce expert medical evidence to show that the physician failed to make the proper disclosures.[20]

During the last two decades, the Kansas Supreme Court has led many other courts in the refinement of the doctrine of informed consent. The former claims of battery, wherein the physician was accused of performing a procedure without authorization of the patient, are rare. Now many plaintiffs add an allegation of lack of informed consent to a claim of negligence in the performance of a procedure. Frequently, the plaintiff acknowledges that there was a consent to do something, but asserts that there was a failure to disclose all of the adverse potential risks of a procedure, which, if known, might have induced the patient to withhold his authorization.

In order to recover, the patient must establish that if he had been informed of the risks and hazards of the procedure, he would not have submitted to it. This determination has to be made objectively. The law recognizes that if a complete disclosure of all facts, diagnoses, and possible consequences would endanger the recovery of the patient because of his existing physical or mental condition, the physician is authorized to withhold such information.

Statutory Changes

In 1976, responding to the nationwide problems caused by the frequency of malpractice cases and the increased amount of settlements and awards, the Kansas Legislature passed a package of legislation upon the subject. The lack of availability of insurance was solved by the Kansas Health Care Provider's Insurance Availability Act, K.S.A. 1978 Supp. 40-4301 *et seq.* This statute creates a state fund under the supervision of the Kansas Insurance Commissioner for payment of all claims exceeding $100,000, and requires all health care providers to maintain such insurance as a condition of licensure. The constitutionality of the mandatory requirement for insurance was upheld in *Schneider v. Liggett.*[21]

Another significant legislative change was the reduction of time in which an action could be maintained for medical malpractice. The law as it previously existed permitted, under certain circumstances, a claim to be filed up to 22 years after the date of the alleged negligent act in some cases involving minors or incapacitated persons. K.S.A. 1978 Supp. 60-513 now provides that a suit will normally be brought within two years unless the fact of injury is not reasonably ascertainable until some time after the initial act; then the period of limitation shall not commence until the fact of injury becomes reasonably ascertainable to the injured party, but in no event shall such an action be commenced more than four years beyond the time of the act giving rise to the cause of the action. K.S.A. 1978 Supp. 60-515 was also amended to reduce the time during which a minor may bring an action to no more than eight years beyond the time of the act giving rise to the cause of the action.

Other statutory modifications included: the creation of medical malpractice screening panels, K.S.A. 1978 Supp. 65-4901; the provision for immunity for reporting information regarding malpractice to the licensing board, K.S.A. 1978 Supp. 65-2898; a requirement that the Commissioner of Insurance report claims against health care providers, K.S.A. 1978 Supp. 65-4910; providing an exemption from liability for hospitals because of acts of non-employed physicians, K.S.A. 1978 Supp. 65-442; establishing a requirement that attorney's fees charged in malpractice actions be approved by the court, K.S.A. 1978 Supp. 7-121 b; providing that under certain circumstances evidence of payments made to a plaintiff from a collateral source may be admissible in evidence, K.S.A. 1978 Supp. 60-471; amending the Good Samaritan statute to broaden its provisions, K.S.A. 1978 Supp. 65-2891; and providing limited liability under certain circumstances for service by physicians upon medical committees of the Society and hospitals, K.S.A. 1978 Supp. 65-4909.

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Malpractice Procedure

From Letter to Courtroom

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THE EXPERIENCE of being sued by a patient is traumatic for the physician. Pride is involved; being a professional, the physician is offended that the quality of his work has been questioned; and there is the belief that his professional standing will be adversely affected.

Filing and Notification

It is usual practice for the attorney filing a malpractice case to sue everyone involved. If there are several physicians who have seen the patient, the principle usually adopted by the plaintiff's attorney is to sue them all. If the patient's care involved hospitalization, there is a strong likelihood that the hospital will be named as a defendant. One principal reason for this is that until the facts are developed through depositions, interrogatories and other discovery procedures, the responsible party as perceived by the plaintiff's attorney may not be clearly identified. Also, conflicts — large or small — are likely to develop between various defendants, and it is rare when some one of the defendants, intentionally or otherwise, does not give some aid or information that helps the plaintiff against some other defendant. Still another reason for joining as many defendants as possible is that there are likely to be several different insurers involved. One may want to settle — others may not. The plaintiff hopes such conflicts will help him.

Each defendant will probably be represented by a separate attorney. It is sometimes possible for more than one defendant to be represented by the same lawyer, but there are complications in this, and the trend is to hire separate attorneys for each defendant, even if the same insurer covers more than one of the physicians.

Court procedures today favor broad and sweeping allegations with few, if any, specifics set out in the plaintiff's initial pleading. If the suit is in Federal Court, that pleading is called a complaint. If the suit is in State Court, it is a petition. But whatever the name, this document initiates the lawsuit.

Some malpractice cases are filed without warning, but in the majority of cases an attorney, acting for the patient or potential plaintiff, writes to the physician advising that he has been retained to investigate a claim and requesting a copy of the patient's records. Sometimes the attorney will ask for an interview with the physician before filing suit. Response to such a request should be delayed until the physician has notified his insurer and asked for guidance. But, in all events, the physician should notify his insurer immediately upon receipt of such a letter. Good attorneys acquire medical and hospital records and make a good faith investigation and analysis of the facts before they file suit. The conclusions drawn from the investigation may not stand up in court, but at least they represent a genuine effort to get some information before filing a lawsuit.

The risk of being sued for malpractice is so great today that a practicing physician must recognize it as an occupational hazard. Outlined here is information regarding procedure involved and preparation for the courtroom.

Preparation

What procedure should be followed by a physician who is sued for malpractice? On receipt of summons or other suit papers, immediate notification of the insurer is essential. Time is critical, and the importance of immediate notification of the insurer and delivery of papers received cannot be overstressed. However, the physician cannot simply turn the matter over to the insurer and expect to continue practice as usual. Enormous demands will be made on the physician's time, energy, and thoughts in preparation of the defense. Most insurers try to employ attorneys who have experience in the medical-legal field. Under the insurance policy the insurer controls the defense of the case, selects the attorneys, and generally directs the proceedings. Most attorneys

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employed by insurers are glad to work with the physician and to have his counsel and participation. Insurance policies no longer contain a provision requiring the physician's consent for a settlement. This article will not debate the merits or demerits of such a provision, but most policies today do not contain it.

Many plaintiff's attorneys include a claim for punitive damages in their lawsuits. Such a claim is based on the charge that the physician's conduct was gross and more extensive than ordinary negligence, and was of such wrongful character that damages should be awarded as punishment, as distinguished from mere compensation. The rule varies from state to state, but as this article is being written, insurance does not cover punitive damages in Kansas. When a claim for punitive damages is made against a physician, the insurance company writes a letter advising the physician that he is not covered for it and recommending that he obtain his own counsel to defend against the punitive damage claim. Most claims for punitive damages are made for the primary purpose of inserting a wedge for settlement.

The attorney employed by the insurance company will move to get all of the facts. His activities will include acquisition of the physician's and hospital's records and interviews with the physician and other individuals who have significant knowledge, *i.e.* nurses and consultants. He then compiles a file for inspection by another physician who is willing to examine it and submit his opinion on the merits, or lack of them, of the case.

It is the role of the defense counsel to go over all records line by line with the physician defendant. The records must be read as is and not as the physician would like for them to be. Counsel must be certain that there are no unexplained discrepancies on these records. For example, are they all in the same color of ink? If so, why? If not, why not? Are there ten or 15 years of entries on seemingly brand new paper? Any attempt to alter any of the records, if discovered (as it probably will be), will be devastating to the physician. Omissions can be explained, and frequently they can be explained very effectively; changes or alterations will be disastrous.

All medical records, no matter how insignificant they may appear, should be brought into the picture. In conferences between the lawyer and the physician, every document should be evaluated. Every sentence in every chart should be reviewed. Nurses' notes should be studied in detail. Records, correspondence, and hospital charts should be thoughtfully studied. The physician should share his ideas with his attorney, and then let him decide whether they are valuable or immaterial.

Early in the proceedings a decision must be made as to whether the case will be tried to a jury or to a judge. Most physicians prefer trials to the judge; however, most defense attorneys agree that a jury trial is a far more safe, predictable and common sense forum, with a greater likelihood of a verdict in favor of the defendant physician. Contrary to general belief, jurors are less likely to award excessive damages than are judges. If there is a close question of liability, jurors are apt to compromise the award whereas judges, once they find liability, generally impose the full measure of damages.

The physician defendant should not discuss his case with his colleagues. The fact that he has been sued is no disgrace, but an informal discussion rarely includes all of the facts, and opinions given under such circumstances, even supportive ones, are not useful. When all facts are pulled together, the physician and his attorney can jointly select another physician from whom to solicit an opinion. That will produce more meaningful results than will casual conversation.

Discovery Procedures

Aggressive plaintiff's lawyers proceed early in the case to take the defendant physician's discovery deposition. From the plaintiff's point of view, this is frequently one of the most important aspects of the lawsuit. Correspondingly, the defendant physician and his attorney must look upon this deposition as an event that requires thoughtful preparation and detailed analysis.

Many a case has been lost for the physician because of inadvertent statements made during the course of the deposition and because of failure to painstakingly review the records in advance. Such advance review may well be the single most important procedure in the preparation of the defense. The plaintiff's attorney will ask many questions about conversations between physician and patient. The patient will remember everything. The physician must be truthful, but must reflect carefully and recall in every way possible his dealings with this particular patient and everything that was said between them. The physician should not use words such as "routine," because this implies an indifference or lackadaisical approach to the patient's problem. He also should avoid statements such as "I always do this," or use of the word "we" when he means "I." A physician is not a plural entity, and his testimony should be in terms of what "he" does, not what "we" do.

In preparation for the deposition, the physician should review all of his own records and all of the

hospital records. He cannot afford to rely on memory. A good plaintiff's attorney will know the records thoroughly, and the physician must totally familiarize himself with the contents of all records bearing upon the patient's care; otherwise, he will be in trouble.

All people are defensive when questioned about their actions, but it is most unwise to display irritation when questioned by the plaintiff's attorney. Courtesy, no matter how provocative the circumstances may be, is the sign of a professional. The plaintiff's attorney will attempt to pin down the physician to every possible detail with the idea of committing the physician defendant to a theory or an explanation from which he cannot later escape. The physician must be candid and truthful, but he should also remember that medicine is an inexact science and that later reflection or research may develop valid theories or explanations for a course of conduct or conclusion.

Interrogatories — written questions to be answered under oath — are frequently employed by both sides to get information. If the plaintiff serves interrogatories, the lawyer will consult with the physician about the answers to be given.

The physician's attorney will probably also take depositions from the plaintiff and any members of the plaintiff's family who might have significant information. Certainly the plaintiff's medical experts will be deposed. The physician should make suggestions and comments to his lawyer about these procedures. It is advisable for the physician to be present when the plaintiff is deposed.

Summary

All of these procedures are time consuming. There simply cannot be "business as usual." The physician must be available and be willing to help his attorney in every way. Such teamwork can mean the difference between winning and losing.

Medical Malpractice

(Continued from page 508)

Summary

The development of the Kansas Law of Medical Malpractice has generally followed that of other states. While refinements in the law as enunciated by *Tefft v. Wilcox* have occurred during the 110 years since the decision in that case, the fundamental statements of law contained therein are still viable. The most significant modifications have been the restriction in the application of the locality rule, and the developing concept of the physician's obligation to inform a patient of the risk and hazards of a proposed procedure. Statutory changes have generally been limited to providing a statutory mechanism for the solution of the lack of availability of insurance, the shortening of the statute of limitations, and other procedural modifications.

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The Kansas Perspective

Countering Frivolous Malpractice Suits

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PHYSICIANS have begun to fight back in what the Kansas Supreme Court has called the "national medical malpractice crisis."[1] Legislatively, Kansas physicians have helped to bring about such recent changes as: (1) the requirement for judicial approval of attorney's fees in medical malpractice suits;[2] (2) the establishment of medical malpractice screening panels to reduce the number of non-meritorious claims;[3] (3) the establishment of immunity for physicians who render emergency care at the scene of the emergency, except in cases of gross negligence;[4] and (4) the requirement that physicians carry malpractice insurance.[5]

Judicially, however, physicians have met with little success in cases against patients and their attorneys for filing frivolous malpractice claims. In the celebrated 1978 Illinois case of *Berlin v. Nathan*,[6] a radiologist — who claimed he had been wrongfully sued for malpractice — filed a countersuit against the patient, her husband, and her attorney. The patient had sued for $125,000 in the malpractice suit, claiming that the physician had mistakenly diagnosed a chip fracture of the finger as a dislocation of that finger. After the malpractice suit was dismissed, the physician's claim for money damages (for mental distress, costs of defending the malpractice suit, attack on his reputation, and increased malpractice insurance premiums) was determined by a jury in the physician's favor. However, the physician's jury verdict for $8,000 damages at the trial court level was overturned on appeal, because the physician had failed to allege and prove certain key elements of his case. There are no appellete court cases of record in the country where a verdict for the physician has been upheld. (*Editor's note:* Since this article was written, the Nevada Supreme Court has upheld an $85,000 award to orthopedic surgeon Charles F. McCuskey, Jr., M.D. The judgment, including $35,000 in compensatory damages and $50,000 in punitive damages, resulted from an abuse of process suit filed against attorney Samuel T. Bull. A malpractice suit filed against Dr. McCuskey by Bull on behalf of his client had previously been found in Dr. McCuskey's favor. This is believed to be the first physician's countersuit upheld at this judicial level.

Examined herein are the legal requirements and defenses of cases that could be filed by Kansas physicians against patients, their attorneys, and others. Although dealing primarily with malicious prosecution, other possible legal theories are also discussed.

American Medical News, September 5, 1980.) Recently, the Kansas Supreme Court in *Nelson v. Miller*[7] held for the physician by reversing a trial court judgment that had been returned against the physician.

This article examines the legal requirements and defenses of cases that could be filed by Kansas physicians against patients, their attorneys, and others. Although it deals primarily with the lawsuit entitled "malicious prosecution," it summarizes other possible legal theories. Since state laws on these suits vary and since this general subject has been adequately covered in other medical[8] and legal[9] journals, this article focuses on the Kansas physician and the prognosis for success in Kansas courts. It does so in part by comparing the analysis and result of the Illinois case of *Berlin v. Nathan,* with the recent Kansas case of *Nelson v. Miller.*

Nelson v. Miller

The plaintiffs (a man, his wife, and son) in the original malpractice suit had contended that several of the defendant physicians had improperly treated the wife when she was pregnant with her son. They had claimed the undetected fetus was injured during a dilation and curettage and that the decision to do a cesarian section was premature. The plaintiffs had

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sued the responsible medical center and five physicians — including Dr. Nelson, a pediatrician — in the amount of $5,000,000 actual and $10,000,000 punitive damages. The claim against Dr. Nelson had involved the treatment of the son several months after birth. The action against Dr. Nelson was soon dismissed on the basis that the plaintiffs could refile later if discovery revealed a case against him. ("Discovery" is the process prior to trial, including the taking of depositions, in which each side learns the facts of the case.) Later, the trial judge had dismissed the plaintiffs' case against the remaining physicians on the ground that the actions of the physicians were based on medical judgment, thus precluding liability for negligence (malpractice). This judgment in favor of the physicians in the malpractice suit had been affirmed on appeal.

Dr. Nelson then sued the attorneys of the plaintiffs; the original plaintiffs were not named as defendants. This case involved two theories: malicious prosecution and simple negligence. Dr. Nelson attempted discovery through formal requests for admissions, interrogatories (written questions), and requests for production of documents, but these were opposed and never answered by the defendant lawyers. The defendants then moved for dismissal on the grounds that Dr. Nelson had not stated a claim upon which relief could be granted. The district court judge sustained the motion to dismiss. Dr. Nelson appealed to the Kansas Supreme Court which reversed in favor of Dr. Nelson, thus giving him a chance to prove his case to a jury.

Before examining the elements of a justiciable case of malicious prosecution for the physician in Kansas as stated in *Nelson v. Miller,* it is important to note what the *Nelson* case is *not.* It is not a case where a jury verdict for a physician is affirmed. Thus, it is not a case where a physician has been given a chance to prove his or her case at the trial level, so the evidence could be examined on appeal. No evidence has yet been presented to the trial court or jury in *Nelson.* (Contrast this case to the Illinois case of *Berlin v. Nathan,* mentioned above. From *Berlin,* we know what facts and legal procedures do *not* make out a case because a verdict *for* the physician was reversed.) All *Nelson* does for the physician in Kansas is to prescribe the legal rules under which a physician could conceivably prevail. But we have from the case no facts to test the legal principles. Physicians are thus still without a case in the country where the physician wins at trial and the verdict is affirmed on appeal. Only in this kind of case will physicians be able to examine the parties' conduct to ascertain what reprehensible conduct makes a case.

Secondly, the *Nelson* case was one against the attorneys and not the patient (again unlike *Berlin v. Nathan).* However, the Kansas Supreme Court does state the elements of a case against both patient and attorney.

Thirdly, *Nelson* does not present all of the potential legal theories. This article will briefly mention these from a study of other cases.

Even with these limitations, however, *Nelson* is as good as any appellate court case in the country at defining the elements of a case for the physician, and it is clearly a case that favors the medical profession in its attempt to combat frivolous malpractice suits.

Malicious Prosecution

Historically, the claim of malicious prosecution has not been favored by courts for two policy reasons. First is the policy of encouraging the seeking of justice through the courts without fear of retaliatory action. Second is the notion that litigation must end at some point. To allow malicious prosecution suits is simply to foster countersuit after countersuit. Still, the cause of action does exist, and this fact shows that the courts have struggled to achieve a balance between "the social interests in preventing unconscionable suits and in permitting honest assertion of supposed rights."[10] The various elements discussed below are reflective of this balance and of the reluctance on the part of courts to expand any further a successful defendant's right to a retaliatory malicious prosecution claim.

Nelson lists five necessary elements of a malicious prosecution suit. The physician, now the plaintiff, must prove:

(a) That the defendant initiated, continued, or procured civil procedures against the plaintiff (physician).
(b) That the defendant in so doing acted without probable cause.
(c) That the defendant acted with malice — that is, he acted primarily for a purpose other than that of securing the proper adjudication of the claim upon which the proceedings are based.
(d) That the proceeding terminated in favor of the plaintiff (physician).
(e) That the plaintiff (physician) sustained damages.[11]

While these elements might appear on the surface to be simple and easy to prove, closer scrutiny reveals the contrary.

Initiating the Suit. When considering the patient's liability, this first element is clearly met, since the patient would have usually been the main party in interest in the original malpractice suit. The element is more important, however, in bringing in other potential defendents who helped stir up the frivolous

malpractice suit, particularly the patient's lawyer and spouse. In *Nelson,* for example, all of the defendants were lawyers. In explaining this requirement, the Kansas Supreme Court stated that whoever the defendant is — patient, lawyer, spouse, or other — that person must be shown to have been "affirmatively active in the initiation or continuance of the prior action."[12] The patient, by being the named party in the malpractice suit, of course takes an affirmative role. The lawyers take an affirmative, if totally different, role.

A spouse, or even someone else, who conspires with the patient and is active in the bringing of the lawsuit could also be sued. In *Berlin v. Nathan,* the Appellate Court of Illinois reversed the verdict against the patient's husband, along with the other defendants. But the court specifically stated that a husband could be held if he conspires with his wife jointly to commit malicious prosecution. One difficulty in the case against the spouse or another person is the matter of proof of an affirmative role in the lawsuit. For example, if proof that a husband helped incite the wife to sue were mainly communications between the husband and wife, these communications, even if discoverable, might be deemed confidential and privileged and not admissible in court. The same might be said of communications between the spouse and the attorney if an attorney-client relationship existed. Communications with others that tended to prove complicity would be admissible. For example, threats of a suit to the doctor himself might be evidence of a spouse's later affirmative role in the lawsuit. Also, as in the original malpractice suit of *Nelson,* the spouse could take an affirmative role by being a named party plaintiff.

Probable Cause. To be successful in the malicious prosecution lawsuit, the physician must prove that the defendant initiated the malpractice suit "without probable cause." In *Nelson,* the Court stated that probable cause exists when "there is a reasonable ground for suspicion, supported by circumstances sufficiently strong in themselves, to warrant a cautious or prudent man in the belief that the party (the physician) committed the act of which he (the plaintiff in the malpractice suit) is complaining."[13]

Note that whether there was probable cause is measured by both an objective and a subjective test. The objective test asks what a cautious or prudent man in the position of the plaintiff would have believed at the time. The subjective inquiry goes to his actual belief. In either case, whether there was probable cause must be decided by viewing the facts and circumstances as they appeared to the plaintiff in the malpractice case at the time that case was commenced.

In applying such tests, courts must strike a balance. The patient, for example, need not exhaust all sources of information;[14] indeed, the formal discovery proceedings of the lawsuit itself are designed to assist the parties in obtaining the factual details often unknown to the parties when the suit is commenced. Yet, if the facts known to the patient reveal an obvious need of further inquiry, probable cause might be deemed lacking.[15]

A patient who discloses to his attorney all facts known to him, earnestly seeks the attorney's advice, and then relies on that advice will likely be deemed to have had probable cause to sue. The patient does not necessarily exhibit lack of probable cause by being mistaken as to the legal consequences of the physician's act.[16] Yet *Nelson* puts a somewhat heavier burden than usual on the patient who relies on the attorney's advice: he must disclose to the attorney all facts known to him and all that could be learned by diligent effort to acquire information.[17]

What is the probable cause requirement of the attorney? The attorney is in a different position than the patient, since the attorney is presumed to "know the law." Yet the probable cause requirement is countered by the ethical duty of the attorney to represent his client fully and zealously within the bounds of the law.[18] The attorney is not required to prejudge the case. But *Nelson* again lays down a tough rule that should aid physicians in future cases: the attorney may not simply rely on the facts related by his client. He must make a diligent effort to learn additional facts, and if necessary "go elsewhere for information respecting the honesty of the claim or the good faith of the client."[19] Naturally, this duty must be viewed in the context of each particular case. For example, the imminent running of the statute of limitations, necessitating the immediate filing of the suit, might mitigate this investigation requirement.

Malice. The third element of a successful malicious prosecution claim — malice — indicates a condition of the mind. It is not limited, however, to personal hatred, spite, or revenge. Malice exists when the malpractice action was instituted for any wrongful motive, that is, a motive other than bringing a party to justice or securing the proper adjudication of a claim. Nor must malice be proved directly; it can be inferred by facts and circumstances surrounding the case.

The Kansas Supreme Court in *Nelson* provided some illustrations[20] of situations where proceedings were initiated for improper purposes. One is where the plaintiff in the malpractice suit knows his claim is

non-meritorius. Such would be the case if it is based on manufactured or perjured testimony. A second case is one begun primarily because of hostility or ill will, which is malice in its literal sense. Suing merely to harass the doctor demonstrates malice. Likewise, a suit brought to force a settlement that has no relation to the merits of the claim is a malicious suit. Often called "nuisance suits," they are indicated, for example, by a gross disparity between the original injury to the patient and the amount of damages sought. The malpractice suit of *Berlin v. Nathan,* for example, was one asking for $125,000 for failing to diagnose a chip fracture in the finger. A jury might infer malice in such a case, because the treatment prescribed for the improperly diagnosed dislocation was the same as would have been prescribed for the chip had it been so diagnosed.

The malice element and the probable cause element are related. Malice may be inferred from the absence of probable cause, but this is a question for the jury.

In a case by a physician against the patient's attorney, malice is still a requirement. The attorney's malice would perhaps less often be shown by ill will or hatred since the attorney has not been involved in the medical diagnosis and treatment aspects of the case. But the other illustrations of malice given above are clearly applicable to the attorney as well. Bringing nuisance suits to force settlements or bringing obviously non-meritorious suits demonstrate malice on the part of the attorney. Yet a related claim in *Berlin v. Nathan* failed. There, Dr. Berlin claimed that the attorney's failure to obtain from another physician prior to suit an opinion on the malpractice question demonstrated malice and lack of probable cause. The court, relying on a Louisiana case,[21] stated that failure to get another opinion is not *per se* a demonstration of malice. Sometimes such evidence cannot be obtained due to time constraints; sometimes it will simply be impossible to find a physician who will support the attorney's theory. In such a case, malice is not necessarily shown. Rather, the attorney and the patient simply filed a poor case and lost. To infer malice in such cases, the Louisiana court had reasoned, would be to brand most attorneys as malicious attorneys.

Termination. The malpractice proceedings must have terminated before the physician can bring a suit in response. If the original action is still pending and undetermined, the countersuit cannot be brought. For example, in *Berlin v. Nathan,* Dr. Berlin filed his countersuit before plaintiff's suit was terminated in his favor. Although the trial judge saw no problem with this suit (in fact allowing such counterclaims arguably promotes judicial economy), the appeals court disagreed: ". . . (If) we were to permit such an action under these circumstances, we would create the incongruous situation of permitting the filing of a suit before the cause of action existed or the statute of limitations commences to run."[22]

Allowing the malicious prosecution suit before termination of the original action would foster even more litigation. Without this rule what would prevent the patient from responding to the physician's malicious prosecution counterclaim with a like counterclaim of his own? And so on. This rule also prevents the evidence of the complaining physician from prejudicing the claim of the plaintiff on his malpractice claim.

"Termination" was defined by the Kansas Supreme Court in *Nelson* as occurring when (1) a competent tribunal adjudicates a claim in favor of the physician, either before or after a trial; (2) the malpractice case plaintiff withdraws the proceedings; or (3) the judge dismisses the proceedings because the plaintiff fails to prosecute his claim. Thus, there need not be a full trial on the merits of the malpractice claim for there to have been a termination.

Must the malpractice action have been filed as such in a court of law, or could malicious prosecution cases arise from other proceedings? The proceeding probably has to have been judicial in nature, but not necessarily a court proceeding.[23] In a 1969 Tennessee case,[24] a board of pharmacy — a state administrative agency — was treated as a judicial body, allowing a malicious prosecution to be based on the complaint to that board.

Damages. The physician must have suffered actual damages as a result of the malpractice suit. In some jurisdictions, this requirement is one to show "special" damages, that is, damages other than the injury and inconvenience which accompany all lawsuits. Illinois enforces this onerous rule. Dr. Berlin in *Berlin v. Nathan,* for example, claimed several specific injuries: (1) attack on his reputation; (2) mental anguish; (3) loss of time spent on the defense; and (4) increased medical malpractice premiums. The Illinois appellate court found the first three of these elements to be "so patently common to all litigation that no discussion is warranted."[25] Furthermore, since all physicians in malpractice cases would likewise suffer with increased premium rates, this injury would not be suffered "specially" by Dr. Berlin as distinct from other physicians.

Fortunately, as shown in *Nelson v. Miller,* Kansas does not apply the special damages rule. The only damage requirement to make a case is that the physician must have sustained damages over the taxable

costs. These may include harm to reputation, expense in defending himself, emotional distress, and specific pecuniary losses suffered as a result of the malpractice lawsuit. Interestingly, Kansas ordinarily does not allow for the recovery of attorney fees by a successful litigant. Yet, *Nelson* seems to give the physician the chance to recover them in the physician's malicious prosecution action.

Two other points are important on the damages question. First, if the physician can show any of the above kinds of damages, he may have a chance to obtain additional damages in the form of exemplary, or punitive, damages, provided his actual damages are proved. For example, in *Berlin v. Nathan*, Dr. Berlin's jury verdict (before reversal) was for $2,000 compensatory damages and $6,000 punitive damages. On the other hand, the actual (compensatory) damages — such as financial loss or injury to the business, and harm to one's reputation — are often difficult to prove. Mental suffering is even more difficult to prove. These damages would be decided by a jury, but without good evidence on business or reputation loss, the judge might not even allow the question to reach the jury. Attorney fees paid in the original malpractice actions, on the other hand, can be proved with precision.

Related Kinds of Suits

Malicious prosecution is just one of the several types of suits physicians have brought to fight frivolous claims. *Nelson* involved, besides malicious prosecution, a claim that the patient's attorneys were negligent in failing to investigate the claim against Dr. Nelson, and in continuing to prosecute the claim once she learned it was groundless. Both the trial court and the Kansas Supreme Court held that an attorney, in the absence of special circumstances, cannot be held liable for negligence to a person other than his client. One reason for this rule is that there is no contract between the physician and the adversary attorney upon which a negligence claim may be predicated. The duty of an attorney is exclusively to the client, not to third persons. Another reason is the public policy of free access to the courts. If opposing attorneys could be sued in negligence by the prevailing party in a lawsuit, this policy would be subverted.

In *Nelson,* the physician also claimed that he could base his negligence case against the opposing attorney on the attorney's breach of his professional ethical rules. One of those rules, D.R. 7-102(a) of the Canons of the Code of Professional Conduct, prohibits an attorney from asserting groundless claims, or harrassing other persons. Again, the Court held against the physician. An attorney cannot be held liable on the breach of an ethical rule to anyone other than to his client. Naturally, the physician could make a complaint through the bar association and possibly subject the attorney to disciplinary action. But he cannot base a damage claim on the breach by the attorney of a disciplinary rule.

Dr. Berlin of *Berlin v. Nathan* attempted to retaliate with an additional claim called "barratry." Barratry is defined as "the offense of frequently exciting and stirring up quarrels and suits."[26] It is the "frequently" requirement that would hurt a physician in such a claim. A single non-meritorious malpractice suit is not enough to create a case of barratry. *Berlin* states that at least three barratrous acts are necessary to create an action against a barrator. Kansas has had no barratry cases on point.

A physician could bring a suit for libel or slander by claiming that the false statements made in the malpractice suit have injured his character and reputation. To prove a libel or slander case, a physician must prove there was (1) publication (2) to a third party (3) of a false or malicious statement with (4) resulting damages to the physician. This appears to be a viable cause of action for the physician, but the law has historically interposed a privilege defense that would likely defeat the physician in most cases. This defense provides that communications made during the course of judicial proceedings are privileged and cannot be the basis of a libel or slander action. Kansas recognizes liability for libel and slander as well as the defense of privilege in a judicial proceeding.[27]

A last cause of action is called "prima facia tort" — the intentional infliction of harm, resulting in damage, without excuse or justification, by an act that would otherwise be lawful. This theory was approved by one New York Appellate Court in a case[28] where the physician was unable to fit his case into a malicious prosecution framework due to some additional requirements for this cause of action by the State of New York. This case is perhaps unique, however, since the physician in the case in fact had had no contact or association with the patient and so could not have been liable in malpractice. The case might therefore be limited to its facts and to New York. Other causes of action — such as abuse of process, invasion of privacy, and gross and wanton conduct — have met with practically no success in the physician-versus-patient context.[29]

Conclusion

What are the chances of a successful countersuit by a Kansas physician? The best cause of action is

probably malicious prosecution. *Nelson v. Miller* has nicely set out the five elements and, as shown above, the court has relaxed the stiffer rules on malice and special damages found in other states. So, on the one hand, a physician given the right circumstances certainly could recover under the Kansas law for malicious prosecution; on the other hand, it would be a difficult case to prove, and each element must be proved — not one can be omitted. Other potential kinds of claims, such as barratry or slander, will seldom if ever provide relief. In all cases, judges will seek to balance the interest of the patient to free and open access to the courts with the interest of the physician to be free from friviolous malpractice suits. In Kansas, *Nelson v. Miller* tells us that now the physician has at least a chance of success.

Acknowledgment

David B. Brushwood, R.Ph. and candidate for J.D. degree, conducted the legal research for this paper.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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Res Ipsa Loquitur

The Thing (almost) Never Speaks for Itself in Kansas

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RES IPSA LOQUITUR is a legal doctrine that allows the plaintiff to continue with his case despite his inability to present specific acts of negligence on the part of the defendant. The Kansas Supreme Court has allowed the application of this doctrine in only two cases involving medical negligence. The purpose of this paper is threefold: first, to briefly explain the doctrine and its general application to medical negligence cases; second, to review the application of the doctrine in Kansas and point out some inconsistencies in that application; and third, to point out an application of the doctrine that poses a special hazard to the defendant witness.

The Doctrine

The doctrine of *res ipsa loquitur* (meaning "the thing speaks for itself") was the result of an 1863 English case,[1] and this case still serves as the best example of what the doctrine means. A Mr. Byrne was walking on a sidewalk when a barrel of flour rolled out of a warehouse window and struck him. There were no witnesses as to why or how it had fallen. It could have been that a rope holding the barrel had broken, or that an employee of the warehouse — suddenly unavailable — had let it get away.

In order to be successful in a suit for damages as a result of negligence, the plaintiff must prove three elements: (1) He was owed a duty by the defendant; (2) There was a specific act or failure to act on the part of the defendant that violated this duty; and (3) The plaintiff was injured as a result of the defendant's action or inaction. In the Byrne case, the owner of the warehouse clearly owed a duty to pedestrians in the street to keep them from being hit by flour barrels falling out of windows, and the injury to Mr. Byrne was clearly caused by the falling barrel. Thus, the plaintiff had the first and third elements of his case, but there was no specific activity on the part of the defendant that the plaintiff could prove. How the barrel came rolling out the window was a matter of complete conjecture — and conjecture does not lead to "proof by a preponderance of the evidence." To correct this patently unjust situation the Court espoused the doctrine of *res ipsa loquitur*.

The Court said that if a specific negligent act by the defendant could not be shown, the plaintiff could proceed with his case by showing: (1) the event or injury was something which by *common knowledge* is known not to occur except for the presence of a negligent act; (2) the instrumentality causing the injury was under the total control of the defendant; and (3) the plaintiff's own negligence, if any, did not contribute to the injury. In the Byrne case, if the jury felt that it was common knowledge that barrels of flour did not fall from second story windows unless there had been some act of negligence on someone's part, then they could award damages to the plaintiff even though no specific act was ever identified.

***Res Ipsa Loquitur* — "The thing speaks for itself" — has seldom been ruled to be applicable to medical negligence cases in Kansas. Following is an explanation of its general application to such cases; its specific application in Kansas; and a possible hazard for the defendant witness.**

Medical Negligence Cases

Res ipsa loquitur has found little application in medical negligence cases due to the usual failure of the plaintiff to show the *common knowledge* element of the doctrine. The Kansas Supreme Court has said:

> . . . a physician or surgeon is presumed to have carefully and skillfully treated or operated on his patient and there is no presumption of negligence to be indulged from the fact of injury or adverse result by reason of such treatment or operation.
>
> . . . to establish liability in a medical malpractice case there must be proof by expert medical testimony that there was a lack of due care or that approved practice and methods were not followed.[2]

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These words should provide reassurance to the physician who must defend a non-meritorious suit. Much is made of the malpractice crisis, but in Kansas the physician defends from a position of strength. The Kansas Supreme Court has allowed the application of *res ipsa loquitur* in only two cases involving medical negligence. Furthermore, a review of these and subsequent cases leads one to believe that in the future the Court will be even more penurious in its application of the doctrine.

Application in Kansas

The first application was in *Emrie v. Tice*.[3] The patient had presented himself for x-ray treatment of a wart on his ear and following treatment had a severe burn on his scalp, head, and neck. The Court said:

> It must be borne in mind that his is not a case based on unsatisfactory results with respect to the removal of the wart . . . on the upper part of (the patient's) right ear. This is a case involving severe burn of portions of (patient's) head, face, and neck and for the damage alleged to have resulted from the latter.

The *Emrie* case is representative of one of the most common applications of the doctrine in medical negligence cases — the patient who is treated for one part of his body and suffers an injury to another part. He is unable to state the negligent act that caused his injury either because he was rendered unconscious (surgery) or because the modality used is beyond his comprehension (x-ray). Having applied the doctrine in the *Emrie* case, the Supreme Court six years later denied the doctrine in a suit whose factual situation seems remarkably similar. *Rhodes v. DeHaan*[4] concerned a patient who was rendered unconscious for abdominal surgery (duodenal ulcer). In his petition the plaintiff alleged that upon awakening he "suffered intense and disabling pain in the right arm, right shoulder . . . he could not use his right arm . . . and was badly swollen in the area adjacent to the elbow . . . and this area of the arm was black and blue in color." He further alleged permanent disability in his right arm following this occurrence. *Res ipsa loquitur* was held not to apply and the case was distinguished from *Emrie v. Tice* by saying that the *Emrie* case applied to x-ray treatments and the doctrine would not be extended to a surgical case.

Expert Testimony

The only other Kansas case in which the doctrine has been applied is *Voss v. Bridwell*.[5] Before discussing this case it is necessary to explain when expert testimony is required in a medical negligence case. Because of the complexity of medicine the jury must rely on experts in the field to explain and give opinions on the various scientific aspects of the case. However, if the act of the defendant is such that a layman's common knowledge tells a juror that the act is negligent, then expert testimony is not needed. The act of leaving a surgical instrument in an abdomen is a common example of this principle. *Res ipsa loquitur* applies to medical negligence cases when the act of negligence is unknown but the result (injury) is such that common knowledge dictates it would not have occurred but for some (unknown) act of negligence. This is to be distinguished from a situation where the act causing the damage is known and a layman's common knowledge tells one that the act is indeed negligent.

In *Voss v. Bridwell* the evidence showed that an endotracheal tube had been placed in the patient's esophagus, resulting in severe brain damage. The proposed surgery — a mastoid operation — had never been started. In applying the doctrine of *res ipsa loquitur* the Court said:

> Ordinarily a person put under anesthesia is not rendered decerebrate . . . in the absence of negligence.
>
> Upon these facts and under these circumstances a layman would be able to say, as a matter of common knowledge and observation, that the consequences of professional treatment were not such as would ordinarily have followed if due care had been exercised.

In a previous case, *Waddell v. Woods*,[6] the Court said that the doctrine of *res ipsa loquitur* "has no application to the death of a patient under an anesthetic." In distinguishing the *Waddell* case from the *Voss* case the Court seemed to split hairs when it said:

> It must be noted the *Waddell* decision says, "while *under* an anesthetic." It does not say where death is caused from an anesthetic. Usually a patient is placed under anesthetic for the purpose of surgery. The failure of the surgery or combination of the surgery and the anesthetic could cause death and fall within the general rules applicable to medical malpractice cases which prevent application of the doctrine.

The Court seems to imply that the anesthesiologist who had a problem but allowed the operation to proceed would find himself in a more defensible legal position than one who stopped the case before surgery was begun.

The paragraph quoted above and other language in the case lead to the conclusion that the *Voss* decision is based more on the fact that there was an act that common knowledge said was negligent (the tube left in the esophagus) than on the doctrine of *res ipsa loquitur*.

It is apparent from the above cases and others in which the doctrine has been denied that *res ipsa*

loquitur has almost no application in Kansas medical negligence law. At the same time the Court has stated that it intends to rule on the doctrine on a case by case basis. Therefore, plaintiffs' attorneys who are without an expert witness will continue to press for the application of the doctrine at every conceivable opportunity. The defendant witness would do well to be aware of a particularly perverse application of the doctrine that has been allowed in other jurisdictions.

Medical *Res Ipsa Loquitur*

In many states the doctrine can be invoked by the testimony of an expert witness. While a layman's common knowledge may not lead a juror to say that the injury could not have occurred without negligence, the testimony of the expert witness saying, "I don't know what specific act of negligence was committed but I can state based on my training and experience that the damages to the plaintiff could not have occurred unless some negligent act has been committed" can allow the judge to approve the application of the doctrine. This variation of the doctrine has been termed "medical" *res ipsa loquitur*. This term reflects the need for medical common knowledge rather than layman's common knowledge. In a 1944 dissenting opinion, Justice Hoch of the Kansas Supreme Court stated:

> (The plaintiff) might . . . produce expert witnesses who would testify out of wide experience . . . that if the treatment is properly given, according to methods known to all who are trained and skilled in the matter, no such injury ever results.[7]

In more recent cases there has been no allusion to the possibility that Kansas endorses medical *res ipsa loquitur*. In a 1973 case the Court said the doctrine has application " . . . provided a *layman* is able to say as a matter of common knowledge that the results would not have occurred if due care had been exercised."[8] (Emphasis added.)

However, the application has found favor in nearby states. In 1973 the Iowa Supreme Court said:

"We see no reason to say that the common experience required to establish . . . *res ipsa loquitur* may not include the common experience of experts."[9]

A review of case law does not disclose a situation in which the problem of *medical res ipsa loquitur* has been addressed by the Kansas Supreme Court. This would be in keeping with their announced desire to decide the doctrine on a case by case basis. Therefore, one cannot say categorically that medical *res ipsa loquitur* might not have approval in a Kansas medical negligence case.

The Defendant's Trap

There is a serious trap awaiting the defendant's witness in those jurisdictions that recognize the doctrine of medical *res ipsa loquitur*. Everyone who might find himself testifying for the defense as an expert witness should be alert to this problem. *Wilkinson v. Vessey*[10] is a Rhode Island case decided in 1972. It involved injuries sustained in the course of x-ray treatments for a chest lesion. The plaintiff produced no expert witnesses but sought to rely on the doctrine of *res ipsa loquitur*. During the trial the defendant physicians testified that injury would not be *expected* to occur if the treatment were done properly. (Emphasis added.) The Court decided that these statements satisfied the requirement for medical *res ipsa loquitur*. The Court said the testimony of two experts could be interpreted to mean that the injury would not occur unless there was a departure from usual practice. This was sufficient to invoke the doctrine. The judgment for the defendants — rendered after the conclusion of plaintiff's evidence — was set aside, and at the new trial the defendants would have to refute the inference of negligence that had been drawn from their own statements.

In *Carvero v. Franklin General Benefit Society*,[11] a 1950 California case, the defendant anesthesiologist testified that she had given hundreds of anesthetics for tonsillectomies and that death had never before occurred. The Court held that this testimony satisfied the requirement for expert testimony and the plaintiff could proceed on the theory of medical *res ipsa loquitur*.

To the physician it is clear that the defendants in both of these cases were simply trying to show that they and their plaintiff patients had been the victim of fate — an adverse result that occasionally occurs for unknown reasons. Some courts, however, have decided that when a rare result takes place the plaintiff can have a presumption of negligence and the defendant must rebut that presumption. All of us, early in our training, heard the expression "Never say 'never'!" The truism of that expression was never more clearly demonstrated than in these two cases.

In summary, the Kansas Supreme Court has been most sparing in its application of the doctrine of *res ipsa loquitur* to medical negligence cases. One of the two cases in which it has received approval would seem to have been later negated when a similar fact situation dealt with surgery instead of x-ray. In the other case there was a known act of negligence and the application of *res ipsa loquitur* seems superfluous. At the same time the Court's firm commit-

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Physician-Patient Relationship

The Legal Ramifications

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THE PHYSICIAN-PATIENT relationship, once essentially personal and emotional, has evolved with the times to include legal ramifications. These ramifications have been and continue to be defined by numerous court cases and various state and federal regulations, and they now encompass many aspects of patient care.

Right to Accept/Refuse Patient

In the absence of a state or local statute, a physician or surgeon is not legally bound to render professional services to everyone who seeks to engage his professional services. According to the precedent established in *Findlay v. Board of Supervisors of the County of Mohave,* physicians — by definition — are not public servants as are common carriers and innkeepers, and are therefore not obliged by law to serve all who seek their assistance.

However, if a physician or surgeon accepts employment, he is bound by law to exercise a high standard of skill and care at all times and to continue treatment until the physician-patient relationship is terminated. Generally, a physician who is employed for a specific service is under no legal duty to continue treatment once that service is rendered. Consequently, the physician is not liable for abandonment for terminating treatment of a patient after performing that service for which he was originally employed.

Payment

Formerly under English common law, it was universally understood that a physician could not recover for services rendered to a patient except on express contract. On the other hand, a surgeon could legally recover in the courts for services rendered, even in the absence of an express contract.

In the United States, this legal distinction between physician and surgeon has never existed, and it is generally understood that either is entitled to full recovery for the reasonable value of services rendered, if not under express contract, then under an implied agreement between the physician or surgeon and the patient.

A physician attending an unconscious person at the request of a third party, such as a parent or spouse, has the right — according to the legal precedent set forth in *Mayer v. Supreme Lodge* — to recover from the patient, or from the patient's estate should the latter die, a reasonable value of services rendered. If the patient is conscious and agrees with a third party that if a physician is called the third party will pay the fee, the physician can still legally recover full compensation for his services from the patient if he knew nothing of the agreement.

The legal ramifications of the physician-patient relationship are extensive and complex. Nevertheless, the underlying criteria in the resolution of litigation are based on common sense. A concern for the best interests of the patient is not only essential to ethical medical practice, but also tends to preclude legal problems.

Medical and surgical care is regarded as a necessary family expense which parents are legally bound to furnish for minor children. If parents (or a parent) should fail to furnish medical attention, or neglect to act when medical care is warranted, they are still liable for services rendered to the child in question. Generally, the husband, but in some cases the wife, is liable, although as a general rule the wife is not liable for medical services rendered when the husband is mentally incompetent. (The liability in this case falls upon the incompetent's estate.)

Consent

The relationship between a physician and a patient is based on the consent of both parties. The general rule is that in the absence of emergency or unanticipated conditions, a physician or surgeon must first obtain consent from the patient, if he is competent to

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give it, or someone legally authorized to give consent for the patient, before treating or operating on the patient. Consent for an operation or treatment arises from a contract between the physician and the patient, and is defined by what the parties understand is to be performed.

The theory of informed consent is based on both the patient's right to be reasonably informed of all material elements, including risks that could affect the decision to consent to the procedure, and the physician's duty to inform the patient in a sound, medical fashion.

Consent to surgery may be given in a number of ways. They include: (1) the patient's express consent by oral agreement; (2) the patient's written consent by the signing of a formal permission form; or (3) the patient's implied consent by conduct, such as voluntary submission to the operation or failure to object. The person who gives consent to an operation must know the dangers involved, if any, or the consent does not represent a choice, and thus the agreement is ineffectual. There is a marked divergence of opinion among courts of the various jurisdictions as to what rules are applicable in an informed consent dispute.

In order to ensure that an informed consent is obtained from a patient, the physician must make the necessary disclosures that form the basis of such consent. The physician must disclose to the patient what "a reasonable medical practitioner would disclose under similar circumstances." The extent and nature of disclosures depend on the medical problem as well as upon the patient. It has been suggested that in some cases certain disclosures would only hinder a patient's medical treatment and, in other instances, are considered poor medical practices.

The burden of proof in informed consent disputes lies with the plaintiff who must show with expert medical evidence what a reasonable medical practitioner of the same school, practicing in the same or similar community, and in the same or similar circumstances, would have disclosed about the risks involved in the treatment or in the operation. In other words, the plaintiff must prove that the physician departed from standard disclosure practices.

Severance of Relationship

The relationship between a physician and a patient continues until terminated in one of the modes recognized by law. A physician cannot discharge himself from a case and abrogate responsibility for a patient by simply abandoning the case or staying away from the patient without due notice.

For example, if a surgeon abandons the patient in a critical stage of a disease to a relatively inexperienced substitute surgeon, without reasonable notice to the patient, the surgeon is guilty of negligence and is liable to the patient for any damages caused by this neglect. Reasonable notice constitutes allowing the patient ample time to secure the services of another physician.

The plaintiff must show that the physician's abandonment was the proximate cause of the injury for which redress and damages are sought. A mere showing of negligence on the part of the physician is not sufficient grounds to sustain the abandonment action.

According to legal precedent, a variety of circumstances constitutes abandonment. These include: (1) an express declaration or statement by the physician to the effect that he completely withdraws from the case; (2) the physician's unqualified refusal to further attend to the patient; (3) leaving the patient during or immediately following an operation while the physician's presence is still required; (4) the physician's failure to attend to the patient, despite a promise to do so; (5) the physician's refusal to treat the patient at a certain time or location; or (6) the physician's failure to give proper instructions before discharging the patient.

If a patient does not pay for services rendered by a physician, this action alone does not constitute grounds for abandonment of the patient, if the need for medical assistance still exists. However, if a patient fails to cooperate with a physician, the situation may justify the physician's abandonment of the case. Also, an intervening illness of the physician does not automatically excuse him from the case where there was reasonable opportunity to notify the patient of inability to continue treatment.

In general, a physician who is employed for a specific occasion or service is under no obligation to continue treatment thereafter. Consequently, the physician is not liable for abandonment for ceasing treatment of the patient when the specific service of employment has been completed.

Disclosure of Records

Federal statutes and regulations, as well as state law, define restrictions relative to medical records. Traditionally, many hospitals release such records upon authorization of the patient after informing the treating physician. These are no longer adequate criteria in all cases.

Prime responsibility in this area rests with hospital administrators and medical record librarians; however, physicians should familiarize themselves with the essence of these regulations. This would include an

understanding of the differentiations between such facets of the issue as disclosure *with* vs *without* patient consent; *full* vs *partial* disclosure; and disclosure *with* vs *without* patient identification. Under some circumstances, in the absence of qualifying procedures, even a court subpoena is not adequate basis for disclosure. In addition, under certain circumstances, disclosure may be made without the consent of the patient.

Malpractice

It is the duty of the attending physician or surgeon to bestow on the patient reasonable and ordinary skill, care and diligence, as physicians or surgeons in good standing in the same neighborhood and in the same line of practice have exercised in similar cases. This obligation is not affected by the fact that the physician or surgeon was initially employed by a third party or by the fact that the service rendered is free.

In every medical malpractice action, the defendant's possession of the knowledge and skill ordinarily possessed and exercised in similar situations by members of the same profession in the same field of medicine is clearly the issue. The physician or surgeon is liable to the patient for the failure to exercise reasonable skill and care.

Battery

Unless a person has consented to a surgical operation, the operation on the body of that person is a technical battery or trespass. The courts — in a case where the patient consented to an operation on the right ear, but after being put under anesthetic was found to be suffering a more serious affliction in the left ear, and the surgeon operated on that ear — have held that a trespass had been committed and that the patient was entitled to recover for damages suffered. The extent of the operation does not alter this general rule.

Also, the courts have held that to subject a person to a blood test is an invasion of privacy and may be classified as assault and battery because it involves the insertion of a surgical needle into the body of that person.

A patient's consent previously given may be withdrawn once the examination or treatment has begun. The physician could be charged with assault and battery if the treatment or examination were continued, provided that the physician's withdrawal under medical circumstances then existing did not endanger the life of the patient.

A patient's consent must not be implied, but must be informed consent. The patient has the burden of proof, according to the courts, to prove that the physician failed to disclose particular risks in connection with the contemplated treatment, and that the patient lacked knowledge of the risks and possible adverse effects of the treatment. However, the courts have held that in malpractice cases involving a physician's failure to conform to medical standards in obtaining the patient's consent, the action need not be pleaded as one of assault and battery, since the traditional elements of assault and battery are absent in most malpractice cases based on the physician's failure to make sufficient disclosure.

Extended Liability

A physician or surgeon may be liable for the negligence or faults of an employee or servant — such as an assistant who is working under his direction — for injuries resulting therefrom to a patient. However, a physician or surgeon is not liable for the acts of another over whom he has no control or authority. Thus, the physician or surgeon is not liable for the negligence of other physicians, interns, or nurses left in charge of the patient to administer care that is necessary following the completion of an operation. It is the general rule that a physician acting solely as an anesthetist and not having control of the operation is not liable for the negligence of the operating surgeon.

The courts are divided as to the liability of a surgeon for the negligence of a nurse assisting in the operation. For example, the surgeon is generally held responsible when he delegates to a nurse the performance of duties that are properly the physician's as a part of the operation, even if the nurse is not in the regular employment of the surgeon.

In making determinations as to whether a nurse's particular negligent act should result in liability for the hospital, the courts do not appear to have a structured standard more precise than that of whether the act is classified as administrative or medical in nature. There is substantial agreement among the courts that keeping count of sponges and instruments is an administrative act, and the positioning of the patient on the operating table has been similarly classified. The court's decision largely reflects the specific facts of a case.

The physician may be liable for the negligent acts of other physicians, nurses, or attendants employed by the hospital in which the patient sustains injuries when such servants become the temporary servants of the physician, but not when they are acting in the capacity of hospital employees.

It cannot be stated as a matter of law the exact moment the responsibility of the surgeon begins,

since it is a question for the jury and is governed by the particular circumstances of a case. The surgeon cannot arbitrarily fix the time at which personal responsibility begins inasmuch as his actions determine the time.

Good Samaritan Law

Kansas, like the vast majority of other states, has a good samaritan law (K.S.A. 65-2891).

It states that a physician, dentist, or any practitioner of medicine licensed under the laws of Kansas or any other state, who in good faith renders emergency care or assistance at the scene of an accident without compensation, is not liable for civil damages for such act, other than damages occasioned by a grossly negligent or wanton act or omission in rendering such emergency care. Undoubtedly, one of the main purposes of such an enactment by the Kansas Legislature was to encourage physicians to act as good samaritans by relieving them of the threat of liability resulting from their assistance in emergency or accident situations.

The law also provides that any physician or dentist, licensed under the state of Kansas or any other state to practice medicine or dentistry, may in good faith render emergency care or assistance without compensation to any minor involved in an accident, in a competitive sports mishap, or any other emergency situation without first obtaining consent of the parent, guardian, or the minor injured. Again, it is stipulated that the physician or dentist is not liable for any civil damages, other than damages occasioned by gross negligence or by wanton acts. Emergency care rendered by a physician or dentist within a hospital, a physician's office, a clinic or an emergency room, with or without compensation, is not exempted from civil suits if negligence can be proved by the patient or family.

According to B. Joseph Stetler, former general counsel to the American Medical Association, and Allen Moritz, M.D., Director of the Institute of Pathology at Western Reserve University in Cleveland, Ohio, authors of *Doctor, Patient and the Law:* "No reported cases have been located involving situations where physicians have been sued for rendering their services in an emergency good samaritan situation . . . none of the good samaritan statutes have yet been interpreted or implied by the court at any level, to our knowledge. . . . " Thus, it appears that there have been no such cases involving negligence by a physician at an emergency or accident at any level or in any jurisdiction.

Summary

It is readily apparent that the legal ramifications of the physician-patient relationship are extensive and complex. Nevertheless, it is notable that the underlying criteria in the resolution of most litigation are based on common sense — reasonable attention to patient understanding, prudence in administering patient care and in delegating duties, and regard for the best interests of the patient. These parameters constitute an essential ethic of medical practice and therefore present no conflict, while at the same time tending to preclude legal problems.

Res Ipsa Loquitur

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ment to rule on *res ipsa loquitur* case by case creates some uncertainty, and the expert witness for the defense would do well in his testimony to avoid language from which the Court can rule that the injury in question is so rare that its very presence creates a presumption of negligence.

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Agency Law

The Health Care Provider

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Most physicians have demanding daily schedules. In addition, there is the duty to keep up with new discoveries at every level of medical practice. The state of the art has now added another duty, *i.e.*, the responsibility for supervising the acts of many other individuals because of the law of agency.

Health care providers are liable from many sources of the law. State statutes and federal laws set forth numerous requirements for compliance by the providers. The various and multiple federal and state agencies with their rules, which are quasi-law, add more regulations. The hospital administrator or physician realizes the direct and indirect power of the Joint Commission on Accreditation of Hospitals and the penalties — in terms of time, expense, and legal ramifications — for lack of compliance with its standards.

Constitutional law affords a legal hearing and redress to the medical staff, employees of health care providers, and patients for their grievances under the health care system. The common law or case law with its legal principles of liability has governed a large percentage of medico-legal litigation for many years.

It is apparent from this listing that there are many different branches or sources of law that affect the health care provider. This article concerns itself with the common-law branch of agency as opposed to the law laid down by the administrative arms of the government which are often referred to as agencies and their rulings as agency law.

Agency law as discussed here is that branch of law whereby one person is not negligent himself but is vicariously liable for the wrongful conduct or negligence of another. This legal doctrine of *respondeat superior* (literally, let the master answer) has been expanded by the courts to new theories of recovery that might not have been recognized 15 years ago. Consequently, new duties among hospitals, physicians, and their employees are created. Liabilities for the breach of those duties arise. The physician as an independent contractor or "captain of the ship" is no longer in a sacrosanct position. As nebulous as the connection between the hospital and its staff may seem, it can become the grounds for vicarious liability with a double edge. Both the medical staff and the hospital must realize that they are liable not only for their own acts but also for the acts of those whom they control or should control. In other words, with the control goes the responsibility. This includes the duty to select, teach, and supervise.

Doctrines of vicarious liability and *respondeat superior* are deeply entrenched within the health care delivery system, and passive nonintervention does not preclude liability. Although each health care provider is liable for his own negligent acts, his supervisory physician is also liable. Responsibility and liability accompany the right to control.

Advancements in scientific diagnosis and treatment through the use of sophisticated invasive procedures and modalities have added multiple liabilities for physicians, hospitals, and their employees. Many of these new techniques and devices are used by paramedics so the doctrine of *res ipsa loquitur* (the thing speaks for itself) may be allowed by the courts to facilitate recoveries. Under this doctrine, the burden shifts to the defendant(s) to prove that they were not negligent. If paramedics are involved, such questions arise as to whether they were properly trained or under the supervision and control of an attending physician.

The hospital corporation has a duty, through its trustees, to provide quality medical care via its facilities, employees, and medical staff. Quality medical care is a right of the patient, even though there may be some difficulty in defining quality care. The courts have clearly recognized the duty of hospitals and their medical staffs to provide quality health care. The *Darling*[1] case, in 1965, held that it was the duty of the hospital nursing service to report suspected substandard care on the part of an attending

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physician. Again, in the *Purcell*[2] case, the hospital was held liable because it had been put on notice that a physician was doing incompetent work but refused to take action. Large judgments were handed down against multiple hospitals in the *Nork*[3] case because the hospitals had failed to monitor, through their medical staff committees, the work of an orthopedist. The legal premise for such a ruling is that the medical staff is not independent but is an agent for the hospital corporation and thereby charged with the duty to provide and protect the quality of care given to the patient. This principle of individual liability of the members of the medical staff was later reiterated in the *Corleto*[4] case. A surgical section committee was charged with the duty to monitor the standard of care but was negligent in so doing with a fellow surgeon. They were collectively and individually cited as defendants along with the hospital trustees and administrators for this breach of duty to the plaintiff.

The negligent acts of resident physicians in training have created confusion as to whether the hospital or its supervising staff physician was responsible for the resident's negligence. Each person is individually responsible for his or her own negligent act; however under the doctrine of vicarious liability, the search for a codefendant usually leads to the "deeper pocket" which is more apt to be owned by the hospital or the attending physician.

Medical staff by-laws usually require that all attending physicians be members of the medical staff, although categories may differ according to the extent of hospital privileges. A resident in training may be licensed to practice medicine by the state licensure board without being a member of the hospital medical staff. The resident does not have admitting privileges *per se*, but must be under the control and supervision of an attending physician who is a medical staff member.

When a resident performed a negligent tubal ligation that later resulted in a pregnancy, the court[5] held that physicians who undertook to supervise care in a teaching hospital had a duty to see that procedures were properly performed. Failure to do so would make them liable.

In another case[6] at about the same time, a leg bandage applied too tightly by a resident caused a foot drop. The court held that when a physician could order certain care and treatment for a patient but could not control which particular hospital employee carried out his orders, there was no basis for a finding of a master-servant relationship through which the physician could be held vicariously liable for those not directly under his supervision.

When a duty is delegated to a specific resident, nurse or professional assistant of lesser expertise, the standard of care required is that of the delegator and not the delegatee. The repair of a laceration by a resident will be judged by the level of expertise expected of the delegating physician.

Vicarious liability for the negligent acts of an anesthetist has been a frequent source of litigation. As previously stated, the hospital is not licensed to practice medicine and can do so only through its agents, *i.e.*, physicians. Anesthesia has been held to be the practice of medicine; therefore a nurse anesthetist, even if certified, is acting as the agent of some physician who is licensed to practice medicine in the state. The hospital may employ the nurse but when he or she administers an anesthetic, which is the practice of medicine, that nurse is the delegated agent of a licensed practitioner. If this anesthetist is not the employee of an anesthesiologist or some other physician, it is the surgeon who is responsible for him or her.

As a general rule, a physician is not liable to a hospitalized patient for injuries caused by a nurse in the employ of the hospital. The physician might become liable if the nurse is found to have been his "borrowed servant" or his agent because of his supervising control. Whether this agency relationship exists between them is a question of fact to be determined in each case.

Emergency rooms often employ "moonlighting" physician residents. Many of these residents are well qualified and licensed by the state. Under these circumstances, they are not in training and are responsible for their own acts, as is any practicing physician. At the same time, a principal-agent relationship does exist between the resident and his employer thereby making the employer liable for the acts of the employed physician through vicarious liability.

Physicians who employ physician's assistants must assume the responsibility for the acts of those assistants. K.S.A. 65-2896e. is quite clear that the assistant performs under the direction and supervision of the physician. The physician's assistant shall be identified as such to the patient and all others involved in that patient's care. If the responsible physician is to be temporarily absent, he must designate another physician to assume his responsibility for the acts of the assistant. With this delegated responsibility goes the liability so it behooves the physician to carefully monitor his assistant.

Summary

The very old law of agency is alive and active in

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Hospital and Staff

Working Together for Quality Patient Care

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ULTIMATE ACCOUNTABILITY of the hospital governing board for the quality of medical care provided is a well established principle of health law. The notion of corporate responsibility was first articulated 15 years ago in the *Darling* case from the state of Illinois.[1]

Some states have legislated responsibilities of hospital trusteeship by defining them in their statutes. Indiana law provides that the governing board is the "supreme authority of the hospital," responsible for its management, operation, functioning, and control. The board is responsible for appointment of the members of the medical staff and assignment of clinical privileges in consultation with the physicians. The staff is accountable to the board for the clinical and scientific work of the hospital and for advice regarding professional matters and policies. Michigan has a similar statute. It unequivocally states that the board of trustees is responsible for operation of the hospital, selection of the medical staff, and "the quality of care rendered in the hospital."

These concepts may be a bit disturbing for trustees and physicians; however, the statutes recite no greater duties for trustees than the courts of Kansas require when the question is before them. Suffice it to say that the law places overall responsibility for the quality of professional care squarely on the collective shoulders of the governing board and medical staff.

The Supreme Court of the State of Montana adequately summarized the complexities of the doctrine of corporate responsibility, saying:

> The integration of a modern hospital becomes readily apparent as the various boards, reviewing committees, and designation of privileges are found to rest on a structure designed to control, supervise, and review the work within the hospital. The standards of hospital accreditation, the state licensing regulations, and the (medical staff's) bylaws demonstrate that the medical profession and other responsible authorities regard it as both desirable and feasible that the hospital assume certain responsibility for the care of the patient.[2]

The achievement of this important trust is possible only through a strong working relationship between the medical staff, the governing board, and administration. A continuing spirit of cooperation and mutuality of purpose are essential for this joint venture to provide consistently high quality health care services in the hospital setting.

Under the law, overall responsibility for the quality of patient care rests with the hospital's governing board and medical staff. Current, strictly enforced bylaws facilitate accountability and cooperation in the delivery of quality health care.

The nature of the relationship between individual physicians and the collective participants in the joint venture should be carefully defined and documented. Each physician should be aware of the rights, responsibilities, and accountabilities of a member of the medical staff. This documentation is achieved principally through the staff bylaws, rules, and regulations — perhaps the single most important document relating to this essential function.

Carefully enforced bylaws contribute measurably to ensuring the provision of high quality patient care by requiring peer review and self-governance. Standards of quality assurance are defined specifically for each staff member and for the hospital. The realization that his professional performance is measured by his peers against these standards re-enforces the physician's motivation to provide his patients with the best possible care.

The existence of current bylaws actually protects the physician in all phases of medical practice, in the office as well as in the hospital. The courts say that "a physician must possess that reasonable degree of learning, skill, and experience ordinarily possessed by members of his profession. . . ."[3] He demonstrates his learning, skill, and experience through staff membership, participation in regular review of the work of other physicians, and personal accountability in the peer review process.

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Medical staff bylaws also reflect changing trends in hospital technology and medical science by dealing with new services and the ways in which they are integrated into the hospital and regulated by the staff. As new equipment, techniques and procedures are introduced, the means by which they are approved and implemented are carefully managed by the medical staff and its various committees.

Legal principles of accountability to the patient and assured procedural due process to an aggrieved physician, once novel, now are basic. Archaic medical staff bylaws are breeding ground for difficult legal problems.

The proposition that a trustee's duties include ensuring that the professional competence and work of staff physicians meet a certain standard of quality is a disturbing reality. Bylaws create the framework for referring this important function to the medical staff for counsel and recommendations.

Admittedly, having up-to-date explicit medical staff bylaws, rules, and regulations is an important and desirable aspect of hospital management and quality care control. But, there is only one thing that creates more legal problems for hospitals and physicians than a lack of appropriate bylaws; that is having them, but not following them.

Few circumstances are more incriminating in the collective mind of a jury than setting up hard and fast rules only to digress from them or, worse yet, to completely ignore them. The bylaws, rules, and regulations of the medical staff become self-imposed standards of care. Deviations from them are consistently argued as strong evidence of professional malpractice or neglect.

Therefore, physicians and hospital executives must know and understand the standards of practice spelled out in bylaws, rules and regulations, must keep them up to date, and must observe them. If one fails to observe ones own rules, liability is virtually automatic. The hospital is better off without rules, than to have them and ignore them. The courts have seen this, basically, as the notion of fair play and simple justice. The maker of rules must play by them.

Medical staff bylaws are being frequently challenged, particularly in suits filed by physicians denied staff membership or clinical privileges for which they have applied. For many years, the courts refused to interfere in discretionary staff decisions in private hospitals. They said simply that a private hospital has the right to refuse to appoint a physician to its medical staff, and that refusal is not subject to judicial review.

Recently, however, there is a definite trend toward review of private hospital decisions. This is especially true if the hospital is the only one in the area and, therefore, constitutes a virtual monopoly as the only place in which the aggrieved physician can practice his/her profession in the hospital setting. It is also true where the reason for non-acceptance of the applicant is arbitrary, automatic, or not in accordance with due process.[4]

The courts are inclined to give the hospital governing board the benefit of the doubt. But it is vital that the bylaws include contemporary concepts of due process and fair play in handling physician disputes. It is in the arena of due process that it becomes particularly important that the medical staff follow the letter of its own law and observe its rules assiduously.

The courts have accepted many reasons for which a hospital and its medical staff may refuse clinical privileges. These include professional incompetency; failure to follow hospital rules or regulations; violation of the customary regulation that requires completion of the operative record within 24 hrs of surgery and a complete history and physical examination within 24 hrs of admission; chronic delinquency in completing medical charts; and material disruption of patient services or hospital practice. Other reasons the courts have found for confirming the hospital's refusal of membership or curtailment of privileges include the physician's inability to work with others, personality difficulties, refusal to accept required emergency room rotation, unfavorable letters of recommendation, restriction of privileges at other hospitals, refusal to release information about professional history, refusal to carry malpractice insurance, and lack of space for additional patients.

In documenting the manner in which the application of any practitioner is handled, three important factors should be reflected in the written minutes of the medical staff, its committees, and the governing board: (1) the evaluation of the professional qualifications of the applicant should be based upon reasons related to the mutual objective of a hospital and its medical staff to provide quality medical care; (2) if admission is denied, it should not be based upon any arbitrary or inflexible rule of staff bylaws that would prohibit admission of a practitioner regardless of his experience or professional qualifications; and (3) there must be an opportunity for the applicant to present his side of the case to an appropriate committee of the staff or the board of trustees, and an opportunity for final review by the board.

While private voluntary hospitals have been given

considerable latitude regarding admission of physicians to the staff and control of their privileges, shelter from judicial criticism for arbitrary refusal is rapidly eroding. It behooves health care professionals — physicians, hospital attorneys, hospital executives, and trustees — to assure that all applications are treated fairly and with the exercise of sound discretion by implementing non-discrimination and due process mechanisms through the medical staff bylaws.[5]

The courts have found that lack of membership in the county medical society cannot be used as a basis for excluding an applicant. Also, the failure to complete an AMA-approved training program should not be grounds for refusal. Bylaws containing provisions of this kind, which automatically exclude practitioners by category, may be found invalid.

The role of allied health sciences personnel should be defined in the medical staff bylaws as well. A form of limited staff category for these individuals — psychologists, podiatrists, physician's assistants, nurse clinicians, and others who are not hospital employees — is appropriate. They should not be entitled to vote in the medical staff, hold medical staff office, or possess other parliamentary powers. But there must be a way to evaluate their qualifications and to define the scope of their contact with patients.

Due Process

What are the elements of "due process"? How can fair review and appeal procedures be effectively structured in the bylaws?

The courts have said procedural due process means the aggrieved practitioner is entitled to:

> Reasonable notice of the charges against him;
> A fair opportunity to express his views in the matter; and to
> Have such considerations examined by a panel of fair minded people.

However, the physician is not entitled to a full blown judicial trial.

There must be a notice of the charges against the physician, including a recitation of the reasons for denial of the privileges sought. This should be in writing, be reasonably specific, but need not be in precise detail. The notice of charges should not be expressed in vague, general terms. Specific dates, times, places, and people should be identified whenever possible.

The bylaws must provide for fair opportunity to be heard. The physician should have the right to call his own witnesses and to cross-examine opposing witnesses. He should have access to medical records being discussed and other documentary evidence.

Most courts do not require the right to representation by legal counsel as an element of due process. Granting the review committee discretion to determine whether the physician may be represented by an attorney does not deny due process. The presence of lawyers is not necessary; however, if the hospital has its attorney there, the physician should be permitted to have his, and vice versa.

Also, due process does not always require a hearing prior to suspension of a physician's privileges. The bylaws should provide for summary suspension under extraordinary circumstances, provided the affected physician is given a hearing within a reasonable time. In such an event, there should be an emergency situation involved and a threat to good patient care.

In terms of the quality of the evidence required in hearings, most courts recognize that formal proceedings with all the embellishments of a court trial are not necessary. Any procedure that affords a fair opportunity for the applicant to present his position is sufficient. Hearsay evidence that has rational probative force is acceptable. Generally, the courts will accept "any information upon which responsible parties would reasonably rely in the disposition of important affairs."

Due process proceedings in the bylaws should be structured to demonstrate a reasonably impartial and objective decision making body. Preconceived convictions at the board of trustees level should be avoided if at all possible. A fair hearing should not place the functions of investigating, prosecuting, and judging in a single person. However, the aggrieved physician is not entitled to a panel of outsiders or of physicians who know nothing about the case. And the fact that some members of the appeal committee have been members of the judicial review committee does not require that they be disqualified. In fact, a member of the review committee may have personal differences with the complaining physician. Such problems do not require that member's disqualification.

The medical staff bylaws should require the applicant to release from any civil liability those persons who will be reviewing his application. The application for staff membership and the bylaws should contain the physician's pledge to abide by the medical staff bylaws, rules, and regulations. He should give express authorization for release of information by any other hospital in which he has practiced. He should also release any person furnishing such information from any civil liability for having done so.

These assurances should be included in both the initial and renewal application forms.

Finally, bylaws should not contain flamboyant or grandiose statements of a hospital's or physician's mission. Some courts have been persuaded to lift such language from the bylaws in order to place liability at the doorstep of the hospital. Bylaws that volunteer statements of noble purpose read very well. But they can rise up to haunt the hospital when the plaintiff's attorney brings them into the courtroom suggesting that they establish a standard of care.

Hospital executives and physicians properly interpret such verbiage as their professional aspirations and their highest objectives, elusive as they may be. But patient's counsel may interpret such objectives as established standards of care that all hospitals, except the one in court, routinely achieve. This misuse of bylaws suggests the wisdom of restraint in rhetoric. The purpose of such statements must be explained; they define a level of performance for which we constantly strive, undaunted by the realization that we may never achieve such superlative goals.[6]

Medical staff leadership and hospital executives must remember that bylaws enforcement is even more important than bylaws drafting. The bylaws must be used and relied upon. The hospital executive and chief of staff who take the initiative to maintain good bylaws serve their board, their professional medical staff and, most important, their patients.

The weighty responsibilities of quality care assessment are handled most effectively where the members of the joint venture — staff leadership, administration, and the governing board — work together to promote mutual goals of quality health care and maintenance. The framework of that important cooperative relationship is founded upon current and sensible medical staff bylaws which, together, they fairly enforce.

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Agency Law

(Continued from page 526)

the way that it applies to most health care providers. The doctrines of vicarious liability and *respondeat superior* are deeply entrenched within the health care delivery systems. Medical staffs are no longer a separate entity but rather an integrated part of the hospital complex as a whole. Because of this integration or as a result of it, the hospital corporation as well as the medical staff and its members have been held responsible for the individual acts of member-physicians through failure to properly select, monitor, and regulate. Passive nonintervention no longer ensures nonliability. Even though the paramedic is liable for his own negligent acts, his supervisory physician is also liable. Responsibility and liability still accompany control or right to control.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

A Decalog for Geriatric Medicine

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GERIATRIC MEDICINE during the past ten years has attracted a remarkable increase in interest. Increasing numbers of elderly citizens (defined for our purposes as those older than 64 years) and the cold hard fact that most of them vote have been the major reasons for this interest. Politicians and various people with their ears to the ground often speak of "The Geriatric Imperative: There are many elderly people who must have unmet needs; we must do something for them . . . anything."

The good physician seemingly has always known how to care for elderly patients; however, there is now pressure resulting from the application of the geriatric imperative to medical practice. Unfortunately the research base in geriatrics and gerontology is so meager that there are few good data to answer many of the important questions regarding care of the aged. For instance, it is not known whether elevated blood pressure in the elderly *should* be treated, and if so, how to do it. The following is a decalog for geriatric medicine *(Table I)* rendered in the style of the Ten Commandments and designed to summarize some major and general principles directly relevant to providing medical care to elderly patients.

1. *I am the elderly person who has lived long in this land. The weak of my generation are dead. I am a survivor.*

We tend to think of the elderly person as frail and weak when many are tough and sturdy. Darwin's concept of survival of the fittest can be applied to human beings. One need only consult standard life tables to note how many hardy people survive into the ninth, tenth, and even 11th decades of life.[1]

2. *You shall treat me with the respect my years on this earth and my survivorship warrant.*

Many would say Americans in the latter part of the 20th century do not respect aged members of their society. If television is an accurate reflection of contemporary views and values, it must be admitted that elderly people and the elderly condition do not enjoy a positive image. A major fear of many elderly people is being physically abused by the young, particularly teenagers. Europeans see our American culture as formless and shallow. Relationships, such as those between old and young, are unstructured and casual, not fixed and formalized. In most Asian, and particularly in African societies, being elderly automatically brings stature and respect. In traditional Chinese culture true maturity was not attained until age 61 yrs. In East Africa the most reverent title one human being can use for another is *mzee*, old man.

We are commanded to respect each elderly person because that person has lasted, overcome, persisted,

TABLE I
DECALOG FOR GERIATRIC MEDICINE

1. I am a survivor.	6. Avoid polypharmacy.
2. Treat me with respect.	7. Avoid sedatives.
3. Remember my sabbath.	8. Use digitalis sparingly.
4. Honor your parents.	9. Treat hypertension rarely.
5. You shall not kill.	10. Treat me carefully.

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survived. Certainly physicians and other health care professionals should be among the first to obey this commandment. Few in our society have a better chance to set a good example for others. There is reason to believe that it will become increasingly difficult for the elderly to command respect in the future. Population demographics are such that each year there will be more elderly people in America and they will comprise a larger percentage of the population as a whole. One might observe that it is easy for Asians and Africans to respect their aged; they have relatively few. There were 3 million elderly in America in 1900, there are 23 million now (11% of the population), and there will be about 55 million by the year 2030.

3. *Remember my sabbath, my right to rest and relief from the cares of this world, my retirement.*

Implicit in this is the expectation of a reasonable lifestyle. The physician often has a unique opportunity to help maintain or improve lifestyle for elderly patients. Attention to preservation of sight and hearing so automobile driving may be continued, and assistance with the myriad of support facilities available to the elderly in most communities so nursing home confinement can be avoided are but two of the things a good physician can do.

4. *Honor your father and your mother that you may long endure on the land that the Lord your God is giving you.*

How fascinating and probably ironic is the likelihood that we can set the standards for the care of the elderly tomorrow (when all of us will be elderly) by the care we provide to the elderly today. This commandment is taken directly from the Decalog of Moses and needs no further explanation in the context of medical care of the elderly.

5. *You shall not kill me because you are inept.*

Elderly patients are likely to have several chronic diseases of variable stability. An acute illness in this setting is more difficult to diagnose and more difficult to treat. Medical acumen needs to be sharp and keen. It takes a better and smarter physician to treat an elderly adult than a young adult. Symptoms, signs, and even laboratory indicators are subtle. The margin for error is less. Pneumonia may present without fever and leucocytosis; the only symptom may be confusion; the only signs may be bronchial breath sounds and rales.

Medicating the elderly is fraught with more hazard than most physicians realize. Adverse reactions to drugs increase in direct proportion to age. Hurwitz found 42 reactions to 667 drugs given to patients under 60 yrs (6.3%) and 76 reactions to 493 drugs given to a group older than 60 yrs (15.4%). Digitalis caused more adverse reactions in the elderly than any other drug, and even when digitalis was excluded the correlation of age to chance of adverse drug reaction was very high and very significant ($r = +0.86$, $p < 0.00087$).[2]

Hospitals and nursing homes deserve special mention. As an elderly farmer once observed, "Hospitals are dangerous places, people die there; nursing homes are worse, almost all of the people who die there are old, like me." There is no question but that elderly people are better off in their own homes than in any other place. Hospitalizations should be as brief as possible and as infrequent as possible. A sprightly retired school teacher once characterized nursing homes as, "Places where you get many medicines . . . frequently, and see a few doctors . . . rarely."

6. *You shall not commit polypharmacy.*

In America the elderly ten per cent or so of the population account for 25 percent of the medicines used. The average elderly person in America is given 13 prescriptions per year.[3] It is not uncommon to encounter an elderly patient who daily takes ten or 12 different medicines, often from different physicians, but occasionally prescribed by a single physician. It would take a very large computer to calculate the effects and interactions of one patient and even five drugs. Particularly at risk is the nursing home patient whose physician prescribes most medicines by telephone and who often receives PRN medications in profusion and on a regular basis.[4]

It is much easier to write another prescription than to take a careful history or examine the patient's body. The tendency to polypharmacy is abetted by the innate human desire to take medicine and by the fact that the pharmacist makes his living selling medicine. In some respects polypharmacy is like adultery; it involves at least three people, feeds on basic human needs, and defies enumeration and statistical analysis.

7. *You shall not use sedatives and tranquilizers except after much thought and then only with great caution.*

The aging brain must function in a basically young world where alertness and quickness of thought are necessary for survival (driving, using power tools, crossing the street). The aged brain is smaller, contains fewer neurons, and functions more slowly than when it was young. All sedatives and tranquilizers compromise alertness. The benefits of tranquilizing drugs even for agitated demented patients must be carefully weighed against the risks. When deemed necessary, a single agent should be employed at the smallest effective dosage. No elderly patient should

receive two or more sedative drugs concurrently. There are many caveats, among them that barbiturates may excite the elderly rather than calm them, and some diazepine drugs have rather long half lives. Of timely interest is the fact that phenothiazines have been implicated in altered thermoregulation in the elderly and can predispose both to hypothermia and hyperthermia.[5, 6]

In general both the number of hours of sleep required per night and the depth of sleep decline with increasing age. It is difficult to justify the use of soporifics at any age, but particularly for the elderly. It is common for even the normal elderly to become confused if awakened in a strange place. It is probably reasonable to assume that most people get enough sleep just as they get enough to eat. It also seems reasonable to assume hours of sleep required are as variable as number of calories required. The safest sleeping medicine is one to two ounces of whiskey as a toddy or a glass of wine such as port just before bedtime.

8. *You shall not use digitalis.*

In the best of situations the therapeutic index of digitalis is probably the lowest of all medicines in common use. The variety of toxicities associated with digitalis poisoning, particularly when arrhythmias are considered, is awesome. The aged heart is often more sensitive to digitalis than the younger heart. Thus, digitalis is the cause of more adverse drug reactions in elderly patients than any other drug or probably any other several drugs. The plasma half life of digoxin is lengthened by up to 40 per cent in elderly people compared with the young according to Ewy *et al.*[7]

The elderly patient with mild to moderate congestive heart failure can usually be treated without hospital admission by using a diuretic such as furosemide, a decreased sodium diet, and bed rest at home. Even if digitalis is used to full therapeutic potential in such a situation, it may later be discontinued without return of the heart failure. For many years it was thought that taking digitalis was a lifetime obligation; however, it has been demonstrated recently that many, particularly the elderly, can stop taking digitalis even after many years' use without ill effect. Relatively slow atrial fibrillation is common in the elderly, and it does not *per se* require treatment with digitalis. According to Dall, maintenance digoxin therapy is probably unnecessary in about 70 per cent of elderly patients.[8]

9. *You shall diagnose hypertension infrequently, treat hypertension rarely.*

One of the great shibboleths of modern medicine is that all patients with elevated blood pressure measurements have the disease we call hypertension and all hypertension should be treated with drugs. To date no controlled study specifically documenting benefits to elderly patients has been published. A very well-designed study is underway in Europe comparing morbidity and mortality in 600 drug-treated elderly patients with 450 placebo-treated elderly controls. It will be some time before this study is completed and published.[9] No one contests that very high diastolic blood pressure should be treated at any age (> 115 mm Hg); however, treatment of systolic hypertension of any level and diastolic blood pressures from 90-115 mm Hg remains controversial in the elderly.

Postural hypotension is very common in the elderly and is accentuated by virtually every antihypertensive drug presently used. Jackson *et al.* elegantly described inappropriate antihypertensive therapy in the elderly in 1976.[10] The incidence of elevated systolic and diastolic blood pressures in the aged is well documented in the classic paper of Arthur Master published in 1958.[11] The data are still useful and such a study of thousands of untreated elderly people will not likely be repeated. He found, for example, 32 per cent of healthy white females from 80-84 years of age had diastolic blood pressures of 100 mm Hg or above. It is not difficult to adopt the attitude that "hypertension" is a spent force as far as excess morbidity and mortality in the aged are concerned. Once again, the weaklings are dead; only the fittest have survived. For a concise recent review, the paper by O'Malley and O'Brien is recommended.[12]

10. *You will listen to me attentively; you will examine my body carefully. You will not cause me needless discomfort or pain; you will not place me in needless danger or risk.*

Perhaps summarizing the previous nine commandments, this final one says the elderly patient needs and deserves a sensitive, skillful, thorough, conservative physician. Medical school prepared each of us to be this kind of physician, and geriatric medicine demands it. A clear understanding of pathophysiology and the science of medicine exhibited in a warm and kindly manner is the art of medicine.

Self Assessment Questions

1. Digitalis preparations cause more adverse reactions than other drugs used in the elderly. (T or F)
2. Barbiturates remain the best soporific medication for the elderly. (T or F)
3. There are sound scientific data from controlled clinical trials supporting active drug therapy of all elderly hypertensives. (T or F)
4. In America there is a positive correlation between increasing age and increasing consumption of prescription drugs. (T or F)
5. At present the elderly (> 64 years) comprise 11 per cent of the USA population. (T or F)

(Answers on page 550)

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Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

The President's Message

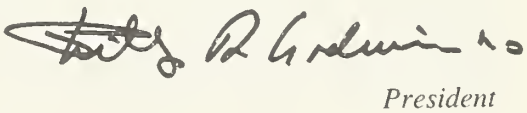
I have always been fascinated by crowds — there is such diversity in the human race. People all look different. They are short and tall, fat and thin, fair and dark, young and old, handsome and ugly. There are no two who look alike. But not only do we look different, we *are* different. Our backgrounds, convictions, aspirations, morals, and expectations are all different. There is also a great diversity within the Kansas Medical Society. As President I have a somewhat unique opportunity as I travel about and correspond with physicians from all over our state. We are urban and rural; we practice solo and in groups; we range from broad general areas of medical care to narrow specialties.

One area of growing diversity is in alternative methods of delivering health care: freestanding surgical centers; health maintenance organizations; independent practice associations; use of physician extenders such as nurse clinicians, midwives, and physician's assistants. Furthermore, there is now a growing movement of consumerism which affects our practices. Even such things as home delivery and lay midwives are emerging. Such changes are hard to accept for many of us inured in more traditional ways of providing health care.

What alternatives are acceptable? Patient outcome is the only justifiable measure for acceptability, for the patient is what it's all about. But predicting the outcome, both short and long term, is difficult, if not impossible. The wrong change could create needless harm to many lives and take many years to correct. But denial of change can be just as detrimental. We cannot stop change, but we can influence it responsibly, and this we must do.



One thing is certain. If we are to continue to insist that we have the health care team, then we will have to be flexible, for many are saying either lead, follow, or get out of the way. If we fail to adapt, we may look like the comic strip boy scout troop leader who is calling to the energetic young boys up ahead, "Hey, slow down, I'm your leader!" Lacking a good measure with which to predict outcome, let's keep our minds open to alternatives while we try to utilize all the unbiased but sound judgment we can muster.



President



Editorial COMMENT

Move Over, Damocles

The attention commanded by numerous changes in the medical scene tends to obscure some changes of serious import which, by their nature, are more readily ignored. It seems appropriate, however, in an issue devoted to medicolegal subjects, to consider the matter of medical fallibility.

This necessitates a little background speculation on the function of the medical body and mind. We assume for this purpose that a sense of responsibility is probably the most fundamental — and necessary — characteristic of the medical practitioner. Of course, it can be said that this quality in itself is not the exclusive property of the physician since it applies equally to the serious practitioners of any legitimate discipline. In medical practice, however, the peculiar quality that sets it apart is due to the object and objective — the patient and his care. Any ennobling nature and of medical service derives from the character of the service more than the practitioner.

If, however, a sense of responsibility of formidable proportions is necessary for medical function, it carries its own sustenance — a profound sense of rectitude. The physician operates from a conviction that his purpose is eminently justified, his efforts are therefore ordained and, all in all, he is deserving of respect both inside and outside of the profession. This attitude can be a necessary support in times of stress but can also be an insulation from some of the social realities and may be interpreted by the public as arrogance. The effectiveness of the medical profession (individually and collectively) depends largely upon how closely the physician's self-image and the patient's view can be brought into common focus.

As the physician embarks on his career, his ability to qualify for his role requires the presence of these qualities in at least embryonic form with an adequate postnatal maturation bringing them to a functional state. The most successful period of any physician's career is in the premedical days. The lives he saves, the secrets he discovers, the miracles he accomplishes are frequent and undiluted — flavored with a seemly modesty as befits the anticipatory fantasy. Medical school is a process of enclosing this fantasy world by exposing the student to the practicalities of medical life as he observes the processes that will utilize his sense of responsibility — and confound his self-esteem. Medical education is a process of applying professional substance to the skeleton of rectitude and activating it with the enzyme of responsibility. But it is also a beginning of awareness that, despite vigilant dedication to principle and technical capability, the opportunities for error or failure are incessant.

Confronted with this disquieting aspect of the noble effort, the physician has the choice of professional paralysis (that is, the least threatening use of his expertise) or devising a code of function that accommodates to this fact of medical life. Traditionally, the latter course has been manifested by a degree of dedication and effect that has been acceptable to the public with only occasional recourse to claims of damage. The physician considered the provision of malpractice insurance to be sufficient external acknowledgment of the possibility of error, this being looked upon not only as a means of buffering himself against the litigious process but as a gesture of consideration to the public as well.

If one asks a physician, then, if he is infallible, he will most certainly say "No" but mean, in his own interpretation, "Yes," since he considers his dedication, preparation, and sincerity of intent adequate neutralization of those uncertainties of medical reality that may leap out at him unexpectedly.

So the distressing and, to the physician, unjust explosion of malpractice charges in recent years has shaken the profession and produced a crescendo of concern about the nature of medical service. There is more than a little irony in the fact that even as medical liability is taking ominous directions, there is increasing public demand for reliance on those of lesser or differing ability. Physician resistance to the inclusion of these individuals and methods is due in

no small degree to the fear that it will mean increasing physician responsibility with increasing physician vulnerability.

In an earlier day when suits for malpractice were less common, they produced considerable dismay in part because of this attitude that the character of medical service should somehow make the physician inviolate. The publicity alone was considered irreparably damaging. The effect of the accusation could never, it was felt, be completely eradicated even if the case was dismissed or found in his favor — witness the front page report of the action and the back page report, if any, of the favorable judgment. The physician's collegues might react with a "but by the grace of God" feeling to the actuality of this ever present risk, but it was easy to remain detached and a general professional defensiveness prompted a belief that the case was probably unjustified.

But the risks of malpractice have greatly increased in recent years not because physicians have suddenly lost their ability or dedication. The complexities of modern practice increase the possibility of iatrogenic damage. The expert and the inept alike are being sued. In order to permit no escapes, plaintiffs' lawyers include everyone remotely connected with the case. Suits are entered on behalf not only of the alleged victim and his immediate family, but others indirectly affected. Even when the patient experiences no direct damage, the physician may be confronted with liability to an affected offspring years later. Those physicians who, by personality or character of practice, establish less positive contact with the patient are at demonstrably greater risk. The inevitable publicity increases speculation by other patients that they may, after all, have suffered some ill effect and seek redress.

It is true that the charge is not equivalent to guilt or conviction, but even the baseless accusation takes its toll. It is reported that less than half of the cases coming to court are settled in the plaintiff's favor, a point plaintiffs' attorneys have made in rebuttal to charges of malice in their filing of cases. It seems a curious rebuttal even if true, since it suggests that if a considerable number of physicians are not being unjustly sued, the plaintiffs must be receiving inadequate service from their lawyers. At any rate, it is a spurious point since it takes no account of those out-of-court settlements which may include those cases of admitted culpability and those in which the settlement is accepted by the defendant as an expedient to avoid the distress and expense of a trial.

Whatever the medical considerations, it must be remembered in terms of perspective that a distinct, perhaps primary, factor in the increase in malpractice litigation in recent years is the current public demand for assurance of safety from all dangers, and an expectation of compensation when damage or even the suggestion of damage results from a product or service. This broad reflection of social attitude has many ramifications beyond the medical. The excessive visibility of the medical profession stems from the same character of medical service that endows it with the particular quality of responsibility noted above, but an accommodation to the problem acceptable to both patient and physician must be supported in part by a resolution of the liability process in general. As in the case of medical care costs, effective control must involve the total economic structure, not just the medical portion.

And so we come to confront the matter of physician responsibility not in terms of sanctity of purpose or high-blown dedication but in terms of fallibility. The plethora of standard-setting bodies is, in itself, an unspoken recognition of our professional fallibility, albeit a professional effort to control it. This is the same fallibility that has always existed, but now it must be considered as an essential element of the medical experience, an understandable expression of the limitations of medical intellect and capability and patient response. We are not dealing just with a statement of limitations embodied in an informed consent release. These remarks are not to be construed as advocacy of a declaration of fallibility on the part of the medical profession but to call to mind some of the factors and evidence that this concept is becoming an operative feature in the social structure and, as with so many other influences in medical practice, will be implemented by others if the medical profession defaults.

It may seem that such concepts as mandatory negotiations and no-fault insurance are expressions of the idea, but they are no more than harbingers. They do remind us, however, that financial considerations are frequently the implementing factor in producing social movement, and that an inherent feature in any system of recognized fallibility would require a concomitant code of compensation. We can anticipate the argument that such would bring some stability and reason to the monetary aspect of liability actions. Certainly no viable plan can be developed without inclusion of this consideration.

So however much the profession considers iatrogenic damage a necessary evil, however much it abhors acknowledged fallibility (with its implication for even more liability actions), the profession must give credence to the signs, arrive at an acceptable position (before being handed an unacceptable one), and be prepared to hold it. — *D.E.G.*

The Menninger Foundation

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Quality Assurance for Psychiatric and Substance Abuse Facilities with Joint Commission on Accreditation of Hospitals, December 11–12, 1980

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DIVISION OF HEALTH CARE OUTREACH AND CONTINUING EDUCATION

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GARDEN CITY — St. Catherine's Hospital
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December 13, 1980 and May 30, 1981

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Content: Orthopedic diagnosis, treatment, and related sciences are discussed in informal case-centered sessions.

Registration Fee: $20.00 per session.

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January 21, 1981

CHANUTE — S.E. Kansas Regional Health Education Center
February 25, 1981

COFFEYVILLE — Coffeyville Memorial Hospital
March 25, 1981

PITTSBURG — Mt. Carmel Medical Center
April 8, 1981

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May 27, 1981

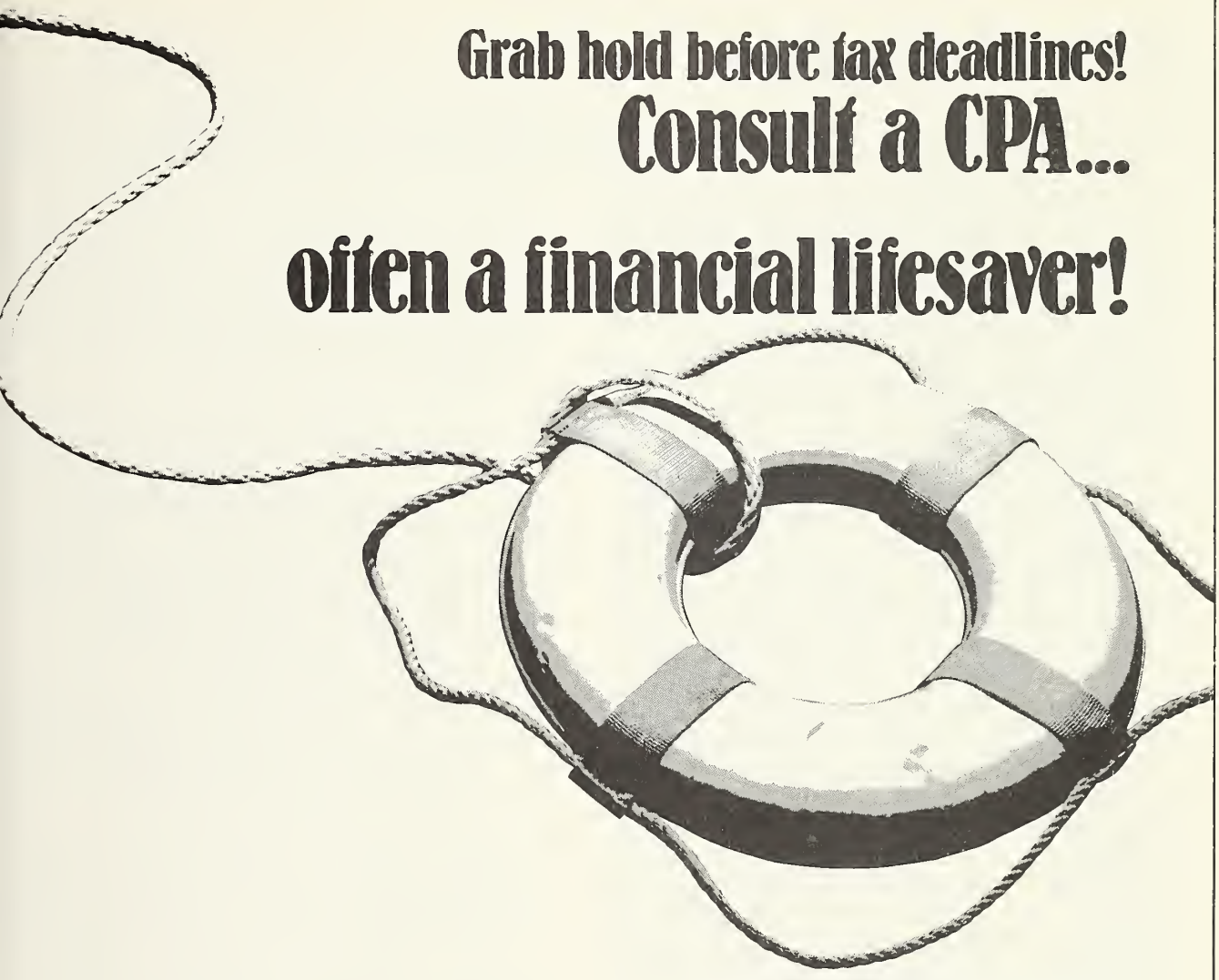
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Registration Fee: $20.00

Accreditation:
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Council Meeting

Report of Meeting Held September 14, 1980

A meeting of the Council was held on Sunday, September 14, 1980 at the Best Western Plaza Inn, Junction City, beginning at 10:00 A.M. Present were: Drs. Phillip A. Godwin, President, Lawrence; Lewis G. Allen, Shawnee Mission; K. M. Boese, Manhattan; Dean T. Collins, Topeka; Jack R. Cooper, Shawnee Mission; Louis M. Culp, Kansas City; Jimmie A. Gleason, Topeka; Donald D. Goering, Coldwater; H. W. Hiesterman, Quinter; John Neuenschwander, Hoxie; James Ransom, Topeka; Ivan E. Rhodes,Wichita; Alex Scott, Junction City; Stephen J. Smith, Arkansas City; William C. Swisher, Wichita; Max E. Teare, Garden City; William K. Walker, Sedan; Roger Warren, Hanover; Wallace N. Weber, Hays; and Kermit G. Wedel, Minneapolis.

Also present was Mrs. John D. Huff, Kansas City, President, KMS Auxiliary. Also present were Gary Caruthers, Val Braun, and Jerry Slaughter.

The Council took appropriate action on the following items:

Endorsed the *Great American Smokeout* on November 20, 1980. This program is promoted by the American Cancer Society to encourage cigarette smokers to give up the habit for at least that one day. The Kansas Medical Society was asked to provide speakers and encourage distribution of publicity materials, as may be requested.

The following actions of the Executive Committee were approved:

SRS Abortion Controversy. Approved filing a petition for intervention in Attorney General's suit against SRS concerning release of names of physicians performing abortions;

AAMA Meeting Honorarium. Approved an honorarium for $1,000 for Walter Menninger, M.D., as the principal speaker at the American Association of Medical Assistants Annual Convention, in Kansas City;

Sports Day Deficit. Approved a policy that sports day activities at the annual meeting should be self-sustaining, and KMS should provide staff support whenever possible. Directed staff to request clarification of deficit from the 1980 meeting;

I.C. Systems. Directed I.C. Systems to notify prospective clients of KMS policy on interest charges.

Ronald McDonald House Project. Endorsed the project in Kansas City, sponsored by the Children's Oncology Services of Mid America;

Criteria for Contributions.. Adopted the following criteria for financial contributions, donations and membership support:

1. The program or organization must be consistent with the mission of the Kansas Medical Society; that is, ". . . promoting the science and art of medicine and protecting the health of the citizens of this state."
2. If the contribution is solicited for a specific purpose, such purpose shall not be inconsistent with current KMS policies.
3. KMS should receive appropriate recognition for the contribution.
4. No solicitation shall be considered unless it is in written form from an appropriate officer or agent of the organization.
5. Except in extraordinary instances, no contribution shall exceed $500.

Dues Adjustment. Approved same for Drs. R. Hartley, H. Coulter, M. Banks, G. Banks, and D. Doss;

1984 Meeting. Directed staff to contact the Reno County Medical Society concerning the 1984 Annual Meeting in Hutchinson;

MEDICAID Procedures. Approved a concept of performance of certain procedures on an ambulatory basis in the MEDICAID program;

Membership Benefit. Endorsed the Hertz Rental Program for Kansas Medical Society members.

The Council heard an update on MEDISERVE. Forty students are presently participating in the program. The original intent was to solicit contributions from foundations and corporations, but this is impossible until IRS determines the tax status (the study is in its second year). The Kansas Medical Society has contributed to MEDISERVE $20,000 in 1980, but more money is needed to meet obligations this year. MEDISERVE Board has frozen new loans for 1980. The Council approved additional $10,000 for 1980, as an advance contribution for 1983.

The new AMA Code of Ethics was reviewed.

The 1981 budget was approved as printed. KMS dues remain at $150. The Council renamed the building fund as the Building and Maintenance

Fund, and approved changes whereby this fund will be responsible for all expenses associated with the building (such as utilities, custodial services and supplies, insurance, taxes, lawn care, snow removal, repairs, improvements, etc.). The Council also approved staff salary increases ranging 9 per cent to 12 per cent.

The Council acted to establish the Health Insurors Liaison Committee, which will broaden the scope of the former Blue Shield Liaison Committee and will provide liaison with other insurance companies in addition to Blue Cross-Blue Shield. Activities of the former Blue Shield Liaison Committee will be incorporated into the work of the Health Insurors Liaison Committee.

In an update regarding the next legislative session, the Council was advised that the Professional Liability Commission will introduce a legislative proposal on confidentiality of hospital medical staff committee minutes; a bill on family planning, which would exempt physicians from liability for providing pregnancy prevention information and services to minors without parental consent; and that the Society will be asked by the Kansas Chapter of American Academy of Pediatrics to introduce a child-restraint seat belt law.

Dr. Dean Collins announced that a legislative seminar, to discuss the issues of health and legislative interests, is being scheduled for February 6-7, 1981. The seminar will be open by special invitation to 25 legislators and 50 physicians.

Regarding the distribution of UKSM telephone cards, the Council agreed that KMS has no objection to UKSM distributing the cards through their own efforts.

The Council reviewed actions of the Welfare Advisory Committee and noted progress in updating some physician fees for outpatient visits.

Lewis G. Allen, M.D., Council District 3, Johnson County, reported that he would be relocating to Cherokee Village, Arkansas. Dr. Allen was congratulated on his participation in this body during the past years.

After viewing the film, "The Other Doctor," prepared by the American Osteopathic Association, the meeting adjourned.

Join KaMPAC

THE MONTH IN WASHINGTON

In the face of opposition from many in the medical research community, the House has easily approved historic legislation greatly extending Congress' control over the federal medical research effort at the National Institutes of Health (NIH).

The National Cancer and Heart and Lung Institutes, formed during the past decade, are subject to the Congressional authorization procedure, but the rest of NIH has remained unencumbered. As a result, the health subcommittees of the Senate Human Resources Committee and of the House Commerce Committee have not been able to exert the type of oversight and control of NIH activities that are enjoyed by most non-appropriations committees.

Many researchers have feared that authorization authority could lead to more political dabbling in NIH activities and possible restrictions on funding.

In an effort to meet these objections, the House bill allows authorizations to be provided for an additional year automatically if program extensions unavoidably are delayed; and sets up an overall authorization of $100 million to assure that unexpected breakthroughs in research would not be impeded by limits in authorization levels.

Although the appropriations committees have great leeway in voting funds, they are limited by law from exceeding authorization levels established by the other committees in Congress.

The issues raised by the legislation were serious enough for opposition to be registered by four former assistant HEW Secretaries for Health, four previous directors of NIH, the American Association of Medical Colleges and the Association of Professors of Medicine, among others.

However, supporters of the bill, led by Commerce Subcommittee chairman Rep. Henry Waxman (D-Calif.) and ranking GOP member Tim Lee Carter, M.D. (R-Ky.), noted that the Surgeon General and current Assistant Secretary for Health, Julius Richmond, M.D., and Donald Frederickson, M.D., director of the NIH, have indicated their backing of the measure.

But Capitol Hill observers believe there is little chance differences between the Senate and House version of the bill can be ironed out in the rush to recess. Nonetheless, sponsors of the controversial legislation promise they will be back with the same proposal in the next Congress.

* * *

The government has opened a pilot program of requiring drug manufacturers to provide patient package inserts for 10 drugs or classes of drugs. The

purpose is to determine whether such inserts are a good way of informing the public and whether more drugs should be covered.

Pharmacists will provide the leaflets when a prescription is filled initially.

The drugs involved are ampicillins, benzodiazepines, cimetidine, clofibrate, digoxin, methoxsalen, thiazides, phenytoin, propoxyphene, and warfarin.

The three-year program will cost an estimated $21 million a year which will be borne by the manufacturers. Each pamphlet will cost about 18 cents, a cost that will be reflected in the cost of the drug. The Pharmaceutical Manufacturers Association said the federal action is "an important first step" toward the objective of getting meaningful information to the consumer.

The "inserts" will describe what the drug is for, what side effects may occur, and how to take the drug properly to get the most benefit. The leaflets — scheduled to be available in pharmacies by mid-1981 — will also be available to hospitals and nursing homes.

The drugs chosen for the pilot program include some of the most frequently prescribed, including the benzodiazepine category of tranquilizers which numbers Librium and Valium among the familiar name brands. About 16 per cent of all new prescriptions would be affected.

Health and Human Services Secretary Pat Harris told a Washington, D.C. news conference that many of the 6 million cases of adverse drug reaction could be prevented or minimized by giving patients more information about their prescription drugs.

During the evaluation period, the FDA will permit the testing of alternative methods such as having drug information available in a book at the pharmacy.

Under the benzodiazepines, the guideline insert includes both a statement that special precautions apply to the use of the drug in elderly patients and a statement about the risk of the patient developing a dependence on the drug. These drugs should not be used to treat anxiety or tension due to the stress of everyday life, the leaflet says. It cautions against using alcohol while taking the drugs.

Warnings on dependence and use with alcohol and other drugs are also featured in the propoxyphene insert. Advice is given in case of an overdose.

Caution against exposure to sunlight is given for methoxsalen.

Patients taking phenytoin are urged to watch for signs of toxicity. A similar warning of toxicity is included for digoxin.

Clofibrate may increase the risk of developing gallbladder trouble or tumors, according to the leaflet. Regular blood tests are urged.

Warfarin can dangerously interact with other drugs, even aspirin, patients are notified. Physicians should be told of unusual bruising or bleeding.

In a rule published in the Federal Register September 12, FDA set forth the new requirements and published draft guidelines for the ten patient package inserts.

* * *

The Supreme Court has delivered the latest defeat to the controversial drug laetrile as an anti-cancer product by refusing to interfere with a lower court ruling that the drug cannot be "grandfathered" into general use as a cancer treatment.

The Justices left standing a federal appeal court ruling that laetrile did not qualify for an exemption under 1962 Drug Act Amendments.

The high court also left intact the Appeals Court ruling that the privacy rights of cancer patients to choose their treatment were not violated by denial of laetrile.

In June 1979, the Supreme Court upheld the federal government's authority to ban distribution of laetrile, reversing a lower-court decision allowing terminally-ill cancer patients to obtain the substance.

Food and Drug Administration officials say laetrile has no value as a cancer cure, but the National Cancer Institute last July began clinical testing of it on patients.

Twenty-one states have legalized the product.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.



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CONTRAINDICATIONS: Hypersensitivity to aspirin or codeine

WARNINGS:

Drug dependence: Empirin with Codeine can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of this drug and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral, narcotic-containing medications. Like other narcotic-containing medications, the drug is subject to the Federal Controlled Substances Act.

Use in ambulatory patients: Empirin with Codeine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using this drug should be cautioned accordingly.

Interaction with other central nervous system (CNS) depressants: Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) concomitantly with Empirin with Codeine may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Use in pregnancy: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, Empirin with Codeine should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

PRECAUTIONS:

Head injury and increased intracranial pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Allergic: Precautions should be taken in administering salicylates to persons with known allergies; patients with nasal polyps are more likely to be hypersensitive to aspirin.

Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. Empirin with Codeine is given orally. The usual adult dose for Empirin with Codeine No. 2 and No. 3 is one or two tablets every four hours as required. The usual adult dose for Empirin with Codeine No. 4 is one tablet every four hours as required.

DRUG INTERACTIONS: The CNS depressant effects of Empirin with Codeine may be additive with that of other CNS depressants. See WARNINGS.

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Vox Dox

Vox Dox Editor:

I am a multiply-handicapped doctor interested in contacting other handicapped physicians in order to obtain some approximation of the size and characteristics of this population in the United States and Canada. There is currently no organization to provide statistical data on the career patterns of such individuals or to what extent various handicaps affect the physician's ability to remain in active practice. Should any physician become handicapped, such information would be of utmost value in determining the feasibility of remaining in any chosen field and would also provide some indication of the career opportunities available to this unique population.

After determining the size and characteristics of this group, attempts will be made to involve the AMA in the planning process. It is assumed that once the need is demonstrated, the AMA will respond in support.

All physicians, active or inactive, with any type of physical handicap are asked to respond.

FRANK C. ZONDLO, M.D.
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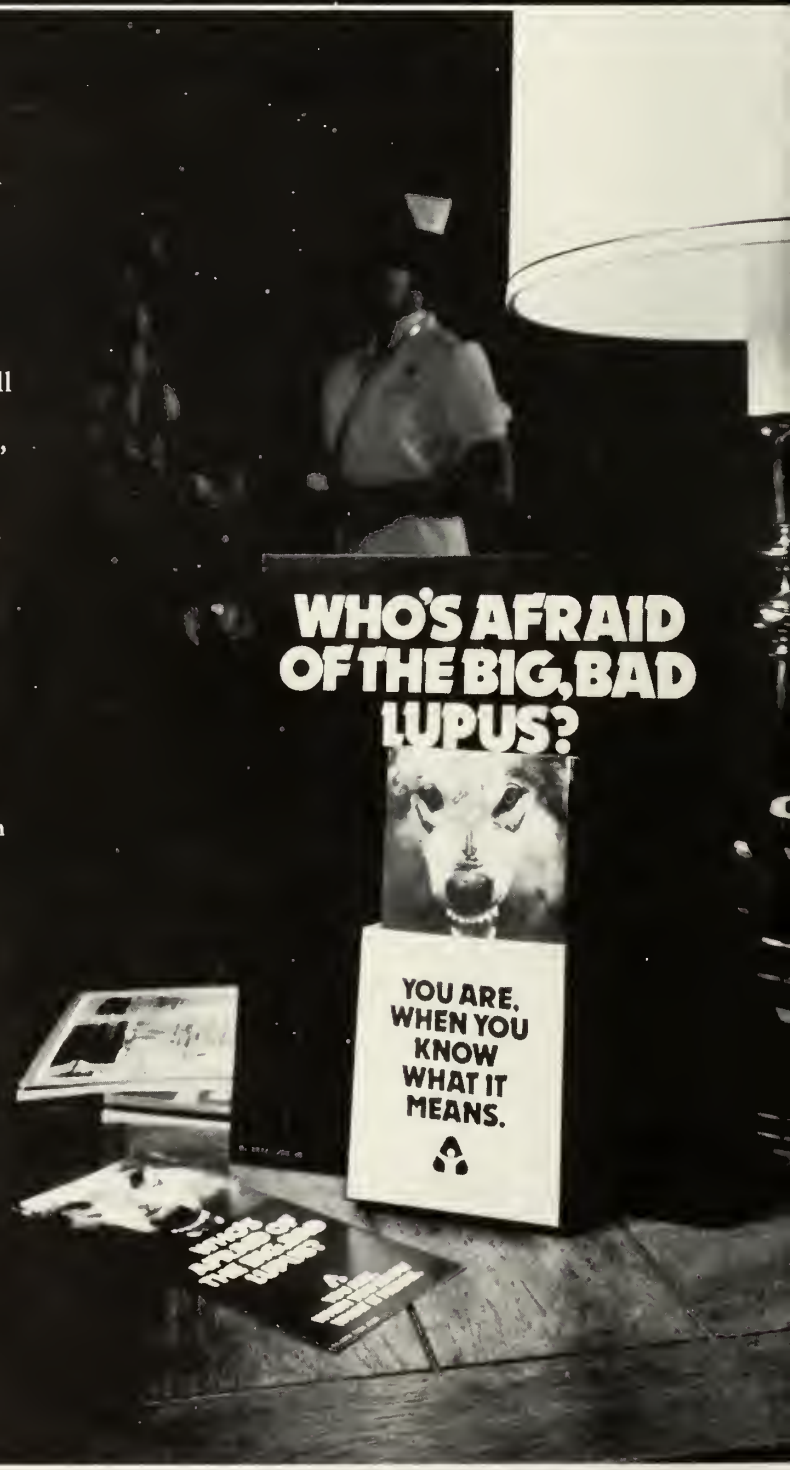
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Lupus is the subject of a new Arthritis Foundation educational campaign.

The campaign theme is: "WHO'S AFRAID OF THE BIG, BAD LUPUS?...YOU ARE WHEN YOU KNOW WHAT IT MEANS." Using a wolf as an attention getter, we will seek to inform the public about the LUPUS problem in the U.S., and the help that is available. Our literature will provide the warning signs for the disease and our consumer ads and broadcast materials will stress early diagnosis and proper medication and therapy, as the only defense.

We urge your participation in this effort. Our new fact-filled lupus brochure, written for the layman, covers such subjects as what is lupus, the diagnosis of lupus, who gets lupus, the pattern of lupus, signs and symptoms, a management/treatment program and prevention guidelines. Simply order the desired quantities from your local Arthritis Foundation Chapter office, or write "Lupus," Arthritis Foundation, 3400 Peachtree Road, NE, Atlanta, Georgia 30326.

With your help, the impact of this lupus educational campaign will be just what the doctor ordered.



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Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librax® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially, increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression. Suicide may be present and protective measures necessary. Variable effects on blood sugar reported rarely in patients receiving [illegible] and oral anticoagulants, causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment; but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and or low residue diets.

ROCHE

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Motrin® vs codeine...

ibuprofen



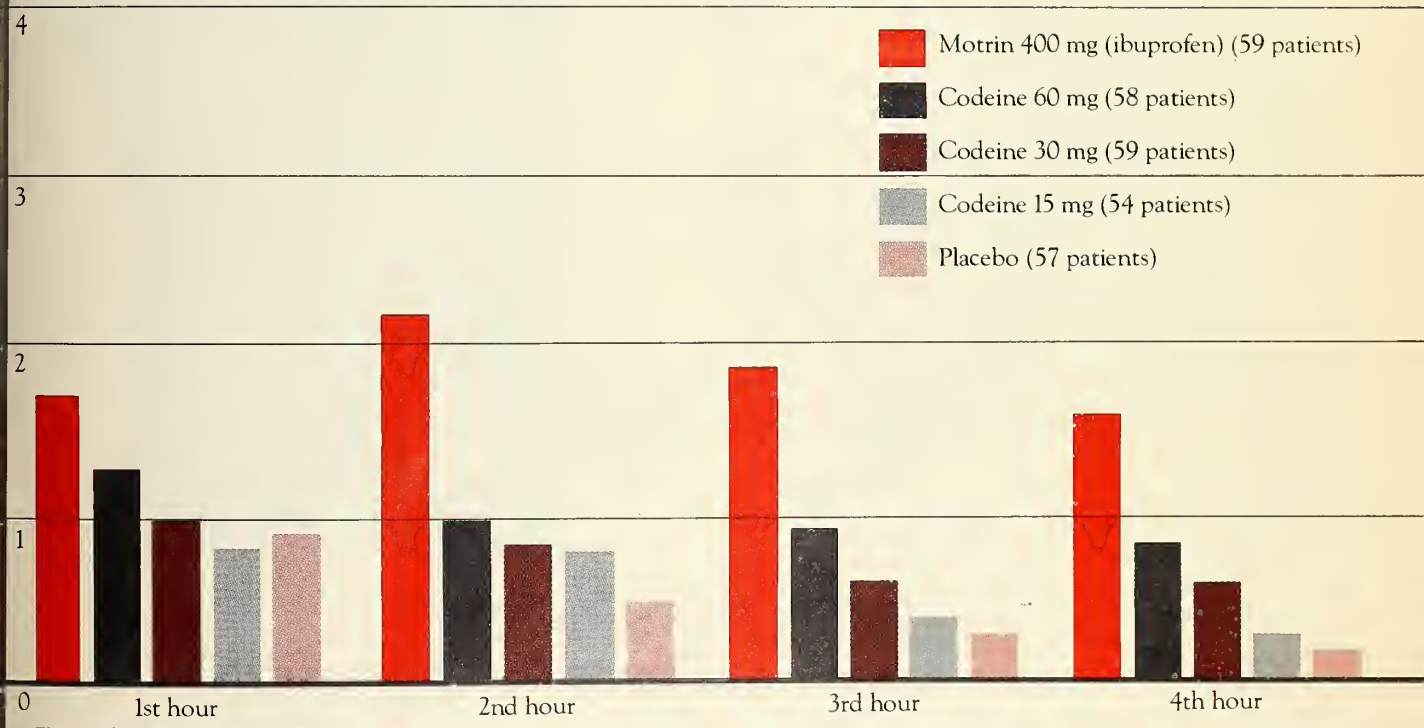
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compare the analgesic effect

Motrin (ibuprofen) 400 mg tablets provided greater relief of pain than codeine in a double-blind, randomized clinical study of 287 patients. *Motrin* was significantly more effective ($p < 0.01$) than codeine 60 mg at the 2-, 3- and 4-hour intervals...significantly more effective ($p < 0.01$) than codeine 30 mg, codeine 15 mg, and placebo at all intervals.

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4 = Excellent relief 3 = Good relief 2 = Fair relief 1 = Poor relief 0 = No relief



Data on file at The Upjohn Company.

One tablet q4-6h prn pain

A well-tolerated, nonarcotic prescription for mild to moderate pain

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ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming • Acts peripherally
- Relieves pain rapidly • Indicated in acute and chronic pain • Well tolerated
- The most common side effect with *Motrin* is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen) now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Relief of mild to moderate pain.

Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.
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Do not exceed 2400 mg per day.

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May 1980

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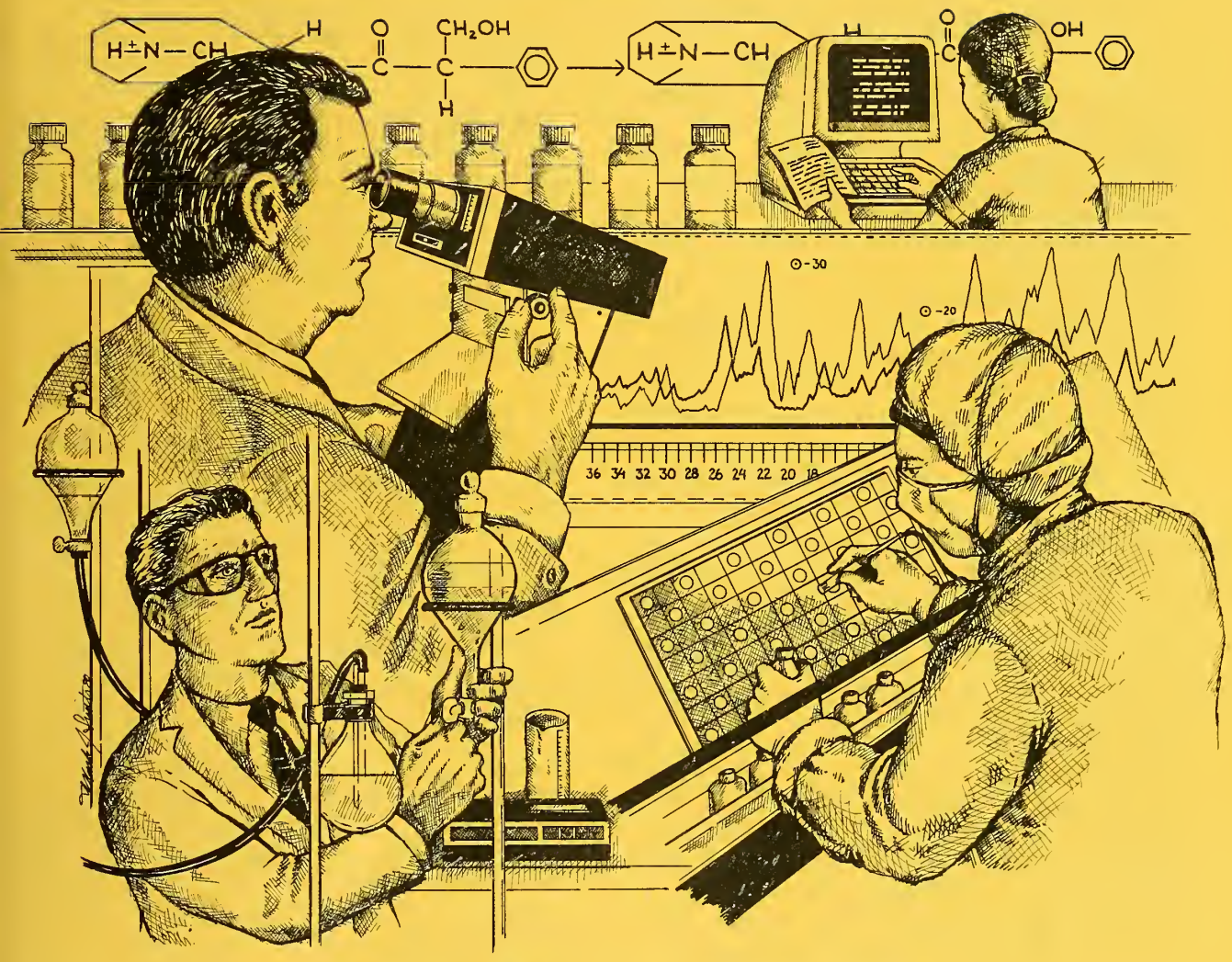
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MENTAL HEALTH "BILL OF RIGHTS" DEFEATED

A proposed provision in the Mental Health Systems Bill that would have provided a "Bill of Rights" for the mental patients has been overwhelmingly defeated on the floor of the Senate.

Senator Robert Morgan (D-NC), with the strong backing of the AMA and the American Psychiatric Association (APA), argued that the private right of action permitted in the provision "will simply serve to encourage frivolous law suits and put the federal courts in the business of reviewing medical decisions on a case-by-case basis."

The measure would have overridden 35 existing state laws and "provide(d) a legal tool to coerce states to spend more money on mental health services," the Senator said in a "Dear Colleague" letter to fellow Senators.

The AMA earlier had dispatched a Legislative Alert urging support of the Morgan Amendment, declaring the Senate Bill "objectionable" as it then stood. The APA feared that court suits by patients would have led to medical treatment decisions by the judiciary, creating serious legal impediments to proper treatment.

The proposed "Bill of Rights" for mental patients, supported by Sen. Edward Kennedy (D-MA), would have given patients the right to refuse treatment and require that their freedom not be unnecessarily restricted, among other provisions.

The overall bill extends and restructures federal aid to community based mental health programs.

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A Decalog for Geriatric Medicine

(Continued from page 534)

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Bactrim™ DS Double Strength Tablets



Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.

Just one tablet b.i.d. for 10 to 14 days

- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
- Distinctive antibacterial action plus wide spectrum helps eradicate recurrent UTI
- Low incidence of bacterial resistance in community practice
- Convenient *b.i.d.* dosage provides day-and-night antibacterial control
- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register, 37*:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Please see back cover.

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XS

Her next attack of cystitis may require

the Bactrim™ 3-system counteratta



ROCHE

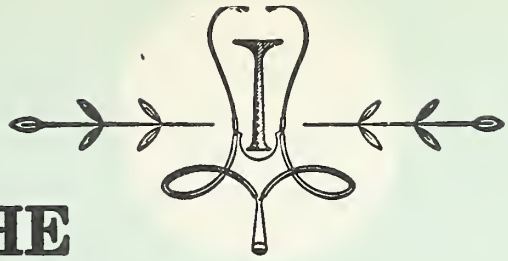
Bactrim has shown high clinical effectiveness in recurrent cystitis as a result of its wide spectrum and distinctive antimicrobial action in the urinary, vaginal and lower intestinal tracts.

The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

Studies have shown that Bactrim acts against *Enterobacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introita colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.



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AMERICAN MEDICAL ASSOCIATION

Principles of MEDICAL ETHICS

Preamble:

The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professionals, and to self. The following Principles adopted by the American Medical Association are not laws, but standards of conduct which define the essentials of honorable behavior for the physician.

I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.

II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception.

III. A physician shall respect the law and also recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.

IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.

V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.

VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environment in which to provide medical services.

VII. A physician shall recognize a responsibility to participate in activities contributing to an improved community.

Adopted by the A.M.A. House of Delegates
July 20-24, 1980

half-life

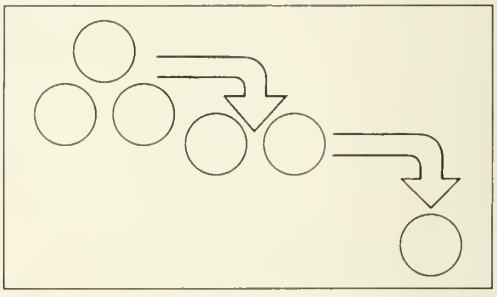
Just one built-in advantage

Ensures smooth therapeutic effect even if a dose is missed The relatively longer half-life of Valium® (diazepam/Roche) has important clinical and pharmacological implications. Steady-state levels generally are reached within 5-7 days with no further accumulation. At this plateau, the patient benefits from the consistent, steady response you expect. Sharp blood level variations, frequently attributed to agents with a short half-life, do not appear with Valium.

Avoids sudden symptom breakthrough Once steady-state levels are achieved, sudden reemergence of symptoms is unlikely. Diazepam and its active metabolites exhibit overlapping half-lives that are advantageous not only during therapy but especially when pharmacologic support is discontinued. Elimination rates are gradual with Valium and thus provide a compatible adjustment interval for the patient. In comparison, blood levels of short-acting agents with inactive metabolites decrease more rapidly and are more likely to be associated with withdrawal symptoms if medication is stopped abruptly.* With Valium unwanted effects other than drowsiness or ataxia are rare. Patients should be cautioned about driving and advised to avoid alcohol.

Tapers naturally; complements gradual dosage reduction at discontinuation When any psychoactive medication is discontinued, it is good medical practice to gradually reduce the dosage. From your own experience you know this is rarely necessary after a short course of Valium therapy, but for patients on extended therapy, gradual reduction of dosage is advisable. This regimen, along with the self-tapering feature of Valium, provides a smooth transition to independent coping.

*Sellers EM: *Drug Metab Rev* 8(1):5-11, 1978



in the management of symptoms of anxiety

Valium®(IV)

diazepam/Roche

2-mg, 5-mg, 10-mg scored tablets

effective therapy through efficient pharmacodynamics

Before prescribing, please see summary of product information on next page

ROCHE®

Valium®(IV)
diazepam/Roche

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy)
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

Editor
The Journal of the Kansas Medical Society
1300 Topeka Avenue
Topeka, KS 66612



American Medical Association Auxiliary
535 No Dearborn St., Chicago, IL 60610

AUXILIARY NEWS

An Open Letter to Kansas Physicians

DOCTOR — Does your spouse belong to the AMA Auxiliary? There are 80,000 Medical Auxiliary members who are involved in community health programs, health care legislation which affects physicians and their families, raising funds for medical students and schools to help ensure the future of quality medical practice, and developing management and organizational skills through leadership training. We are now collecting dues for 1980-81. If not already a member, please ask your spouse to join us. We can do more together.

The Leadership Conference in Chicago was attended by six Kansas Auxiliary members. There were four County Presidents-elect — Pat Elledge, Great Bend; Kay Borkland, Parsons; Jan Chang, Wichita; and Carmen Mimosa, Dodge City — and Betty Moore, State President-elect and myself. We had the privilege of attending some very good workshops on leadership and health programs.

We were privileged to have Marge Smith, AMA Auxiliary North Central Regional Vice President, with us at our Fall Conference in Emporia. She spoke on membership and the AMAA projects and programs. Jean Staab, Nutrition Resource person for the Manhattan School District, discussed the selection of food for good nutritional value. We also got in some exercise when Vickie Ryan, President of Flint Hills Auxiliary, involved us all in aerobic dancing. This was all part of our Shape Up For Life program.

Betty Moore and I have been visiting County Auxiliaries this fall. These auxiliaries are busy with a variety of community and medical projects such as baby-sitting courses, book sales, Christmas sharing cards to benefit AMAERF, scholarships, body pollution learning centers, blood pressure screening programs, and health conferences.

Our congratulations go to three physicians' wives who have just been elected to seats in the Kansas Legislature: Rochelle Chronister (Mrs. Bert), Neodesha, was elected to a second term as state representative; Norma Daniels (Mrs. Robert), Valley Center, won the race for state senator; and Jesse Branson (Mrs. Vernon), Lawrence, was elected state representative. We are proud of them and wish them much success.

I want to thank Jackie Burnett and Dr. David Waxman for writing the Auxiliary News the past two months. Their contributions were very interesting and informative, and I hope you read them.

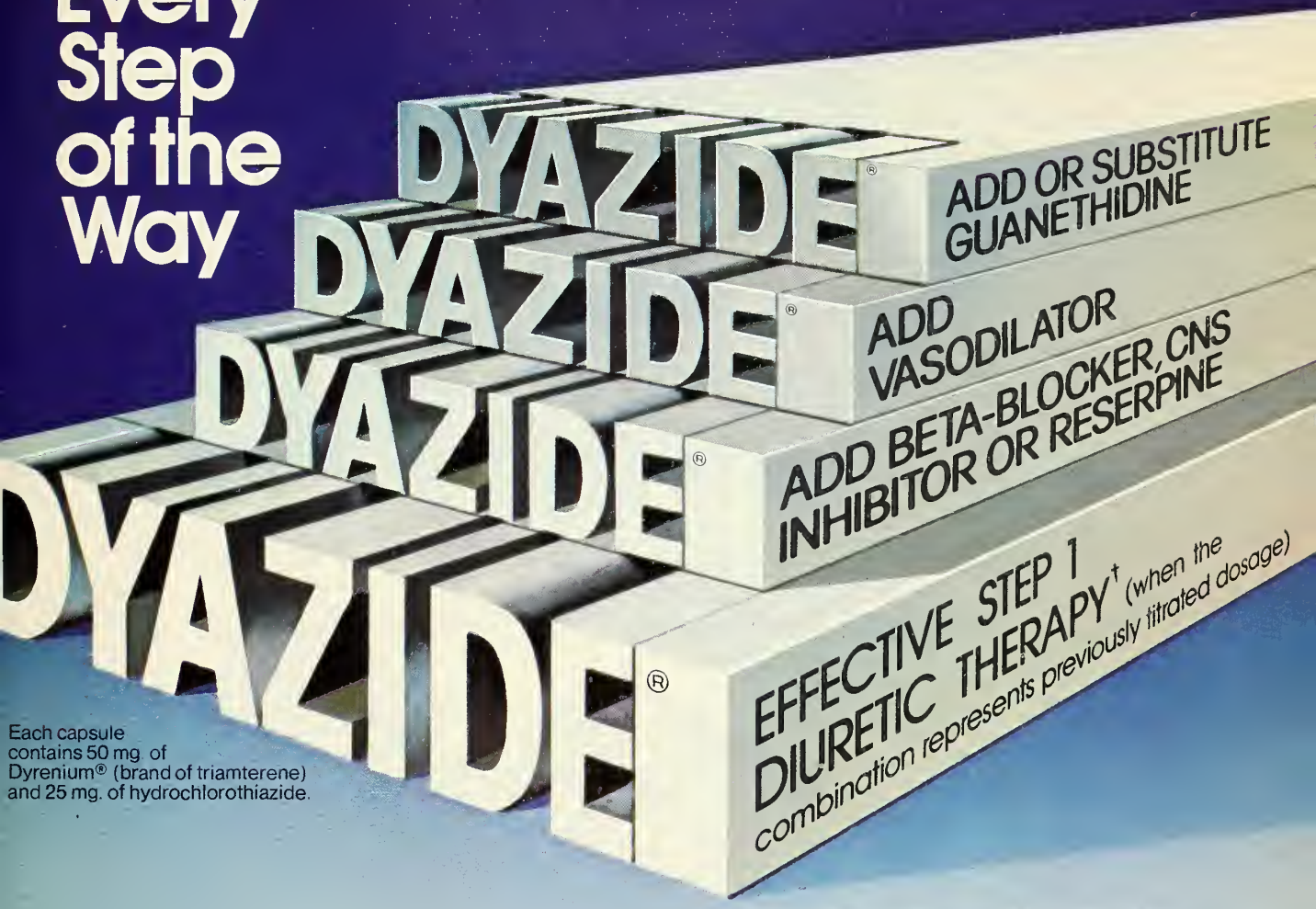
A New Year's resolution we can all make is "Shape Up For Life" and set about starting good nutritional and exercise programs for ourselves.

Merry Christmas and Happy New Year!

Evelyn Huff,
President
Kansas Medical Society Auxiliary

In Hypertension*...When You Need to Conserve K^+

Every Step of the Way



Each capsule contains 50 mg. of Dyrenium® (brand of triamterene) and 25 mg. of hydrochlorothiazide.

†Step 1 usually consists of an initial phase (a diuretic alone), a titration phase (dosage adjustment and/or addition of a K^+ supplement or K^+-sparing agent), and a maintenance phase (a diuretic alone or in combination with a K^+ supplement or K^+-sparing agent).

Serum K^+ and BUN should be checked periodically (see Warnings).

Before prescribing, see complete prescribing information in SK&F Co. literature or *PDR*. A brief summary follows:

* **WARNING**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in postsympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components.

Supplied: Bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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works well in your office...

NEOSPORIN® Ointment

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Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

works just as well in their homes.

- It's effective therapy for abrasions, lacerations, open wounds, primary pyodermas, secondarily infected dermatoses.
- It provides broad-spectrum overlapping antibacterial effectiveness against common susceptible pathogens, including staph and strep.



- It helps prevent topical infections, and treats those that have already started.
- It contains three antibiotics that are rarely used systemically.
- It is convenient to recommend without a prescription.

NEOSPORIN® Ointment—for the office, for the home.
(polymyxin B-bacitracin-neomycin)

Effective • Economical • Convenient • Recommendable

Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

Wellcome

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Symptom: rising administrative costs

Prescription: cost-containing ideas from Southwestern Bell

He's Vice-President for growing Methodist Hospital in Dallas. She's a health care Account Executive for Southwestern Bell.

And whether she or one of her health care team members is reviewing procedures in the admissions area or consulting with hospital administrators, she's one hard-working "problem solver."

Able to dig into your health care facility, study it carefully and emerge with new ideas about how you can use communications to increase efficiencies and contain costs.

For example, after thoroughly analyzing Methodist Hospital operations, Suzanne recommended a special feature Dimension 2000 telephone system.

The results — emergency room attendants receive vital information from doctors faster than ever, patients can easily contact admissions clerks handling their files and the hospital's telephone attendant requirements were cut in half.

To find out how a Southwestern Bell Account Executive can help you solve your health care problems and contain costs, call us today.

Southwestern Bell



ANUSOL-HC

SUPPOSITORIES/CREAM WITH HYDROCORTISONE ACETATE

#1 prescribed hemorrhoidal product

IT WAS NUMBER ONE IN 1959

AND IT STILL IS...

The professional source of *modern* anorectal comfort



ANUSOL-HC® SUPPOSITORIES
Hemorrhoidal Suppositories
ANUSOL-HC® CREAM
Rectal Cream with Hydrocortisone Acetate
Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%, benzyl benzoate, 1.2%; Peruvian balsam, 1.8%, zinc oxide, 11.0%, also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg, bismuth subgallate, 22.5 mg, bismuth resorcin compound, 17.5 mg, benzyl benzoate, 12.0 mg, Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

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As with all anticholinergics, inhibition of lactation may occur.

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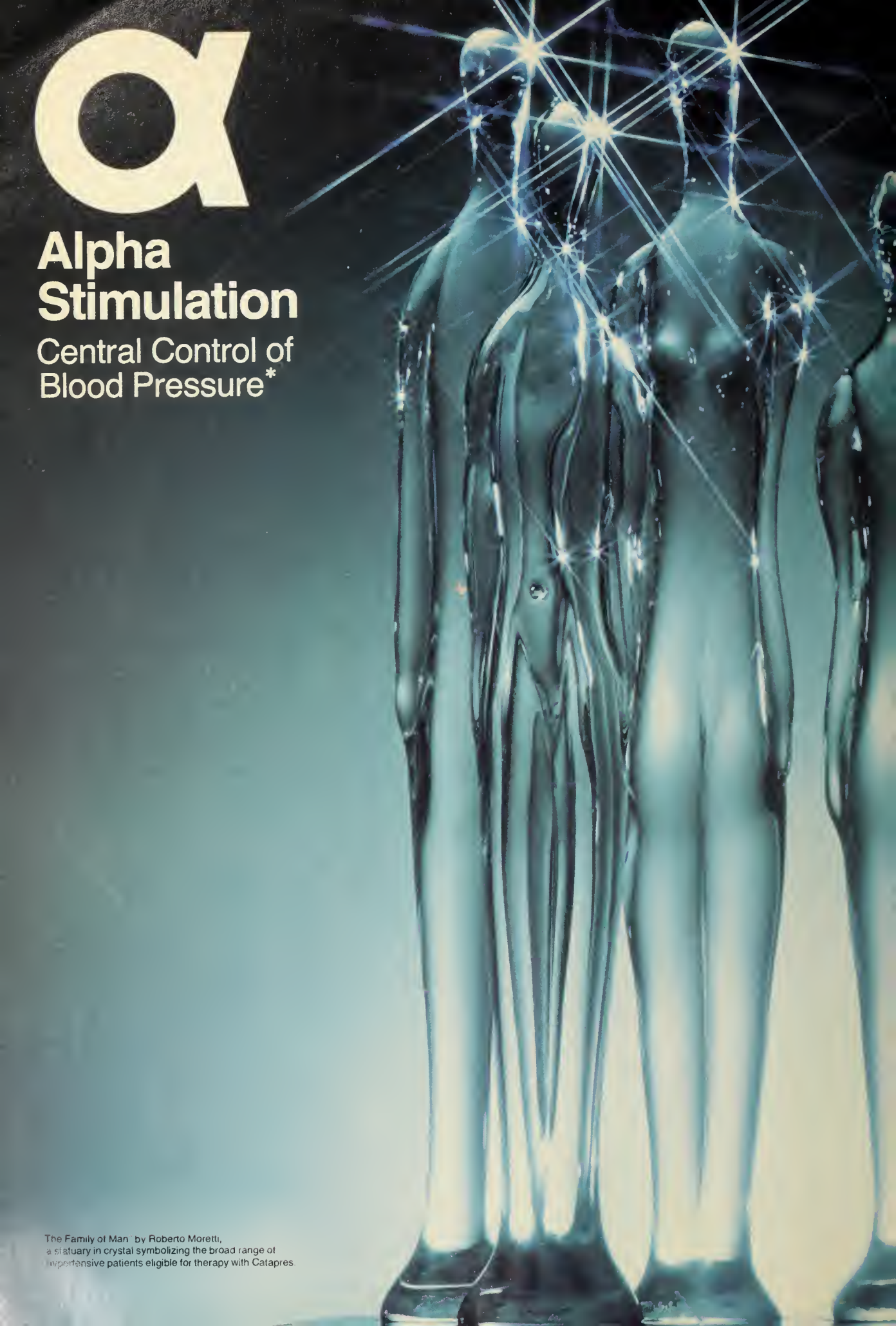
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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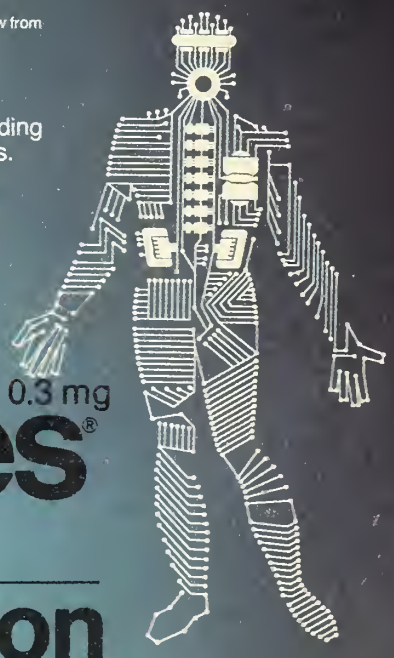
The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

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Please see last page for brief summary, including warnings, precautions, and adverse reactions.

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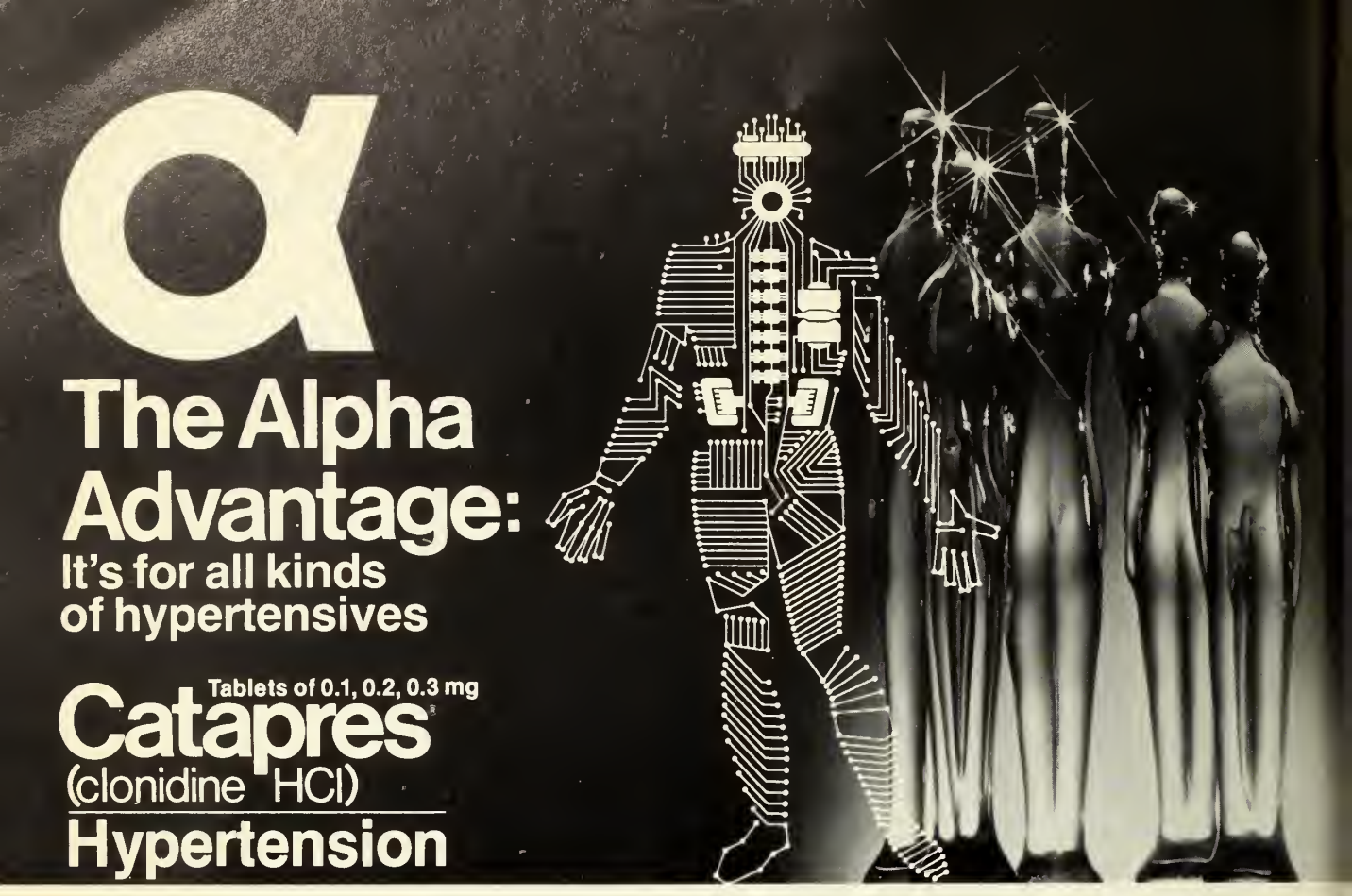


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The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

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Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

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Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

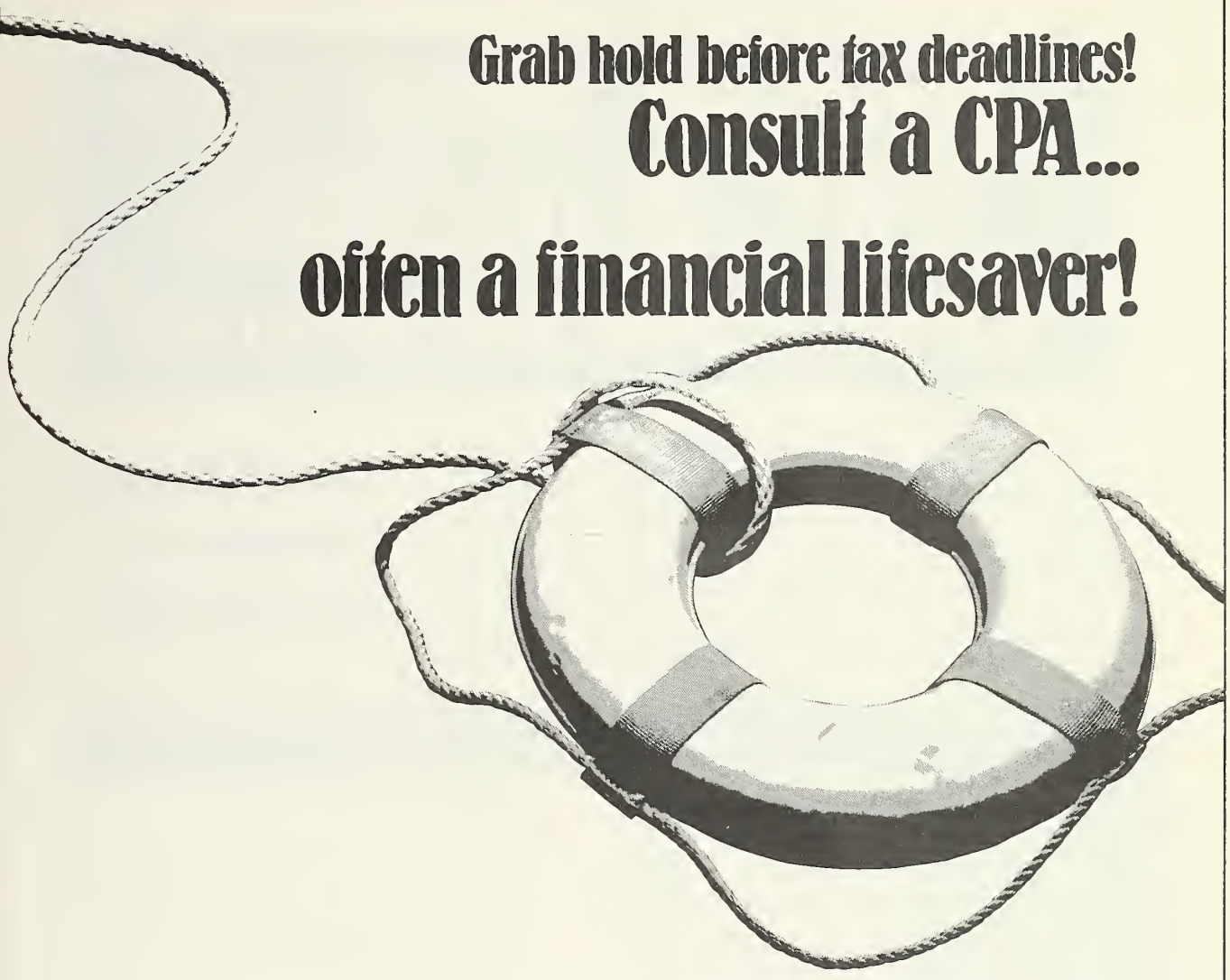
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Scientific ARTICLES

Jogging

A Non-Traumatic Exercise?

CARL K. ZACHARIAS, M.D., *Dodge City, and* H. O. MARSH, M.D.,* *Wichita*

Reported here are the cases of two young athletic individuals who sustained stress fractures of the femoral neck as a result of jogging. It is recommended that attention be paid to complaints of this nature and that adequate investigation be undertaken with x-ray, tomogram, and bone scan as indicated. Diagnosis must be made before the fracture disimpacts.

PHYSICAL FITNESS has been accorded a great deal of attention in our nation during recent years. Manifestations of interest include tennis, racquet ball, swimming, golf, and jogging — to mention only a few. A recent issue of *U.S. News and World Report* summarized this phenomenon with the following statistics: Sixty-three per cent more high school students are involved in interscholastic sports than in 1970, and most of this increase is in women's participation which is up 609 per cent with 2.1 million women now involved in high school athletics. One has only to drive along the road in early morning or late afternoon to observe legions of joggers. They come in all sizes, heights and shapes, and in two sexes. To some degree they must be psychologically anti-social for characteristically they are lonely fig-



Figure 1. AP film one month after original visit. Arrow points to location of fracture which was not diagnosed at this time.

* Associate Clinical Professor of Orthopedics, UKSM-Wichita.

Address reprint requests to Dr. Marsh, P.O. Box 1358, Wichita, KS 67201.



Figure 2. AP x-ray of hip. Arrow points to fracture. Note new bone formation on inferior cortex of femoral neck.



Figure 3. Arrow points to fracture. Further healing of fracture is noted. Note new bone formation on inferior surface of femoral neck.

ures, struggling along the road with a grim, unsmiling countenance gasping for breath.

In any event, jogging is considered a non-traumatic sport that produces only a variety of pulled leg muscles, painful knees, calcaneal bursitis, and a few other allied minor complaints.

Occasionally joggers do experience more serious injuries as evidenced by the following two case reports of femoral neck stress fractures. The patients were healthy athletic young adults.

Case One: In October 1977, a 17-year-old male presented with pain in two areas: (1) anterior aspect, left thigh, just above the knee; and (2) laterally over the greater femoral trochanteric area. The pain was associated with jogging and jumping, and disappeared with rest. X-rays of the hip *(Figure 1)* were negative. The patient was advised to curtail his physical activities.

In November, the patient returned complaining of continued and increased pain. He had not decreased his activities, and pain was still being produced and aggravated by physical activities.

X-rays at this time revealed an undisplaced fracture of the femoral neck *(Figure 2)*. He was then barred from all sports and placed on crutches for two months until late January 1978. At that time he was asymptomatic, the fracture was healed *(Figure 3)*, and the resumption of sports was permitted. There has been no recurrence of symptoms.

Case Two: In May 1979, a 25-year-old female presented with running-associated pain in the left groin, down the anterior aspect of the left thigh, and about the greater trochanteric area. Two weeks earlier the pain had appeared suddenly during jogging, but disappeared with rest. Her routine was to run seven miles/day. All hip motion produced pain. X-rays of the hip were negative *(Figure 4)*.

The patient was placed on crutches and a complete non-weight bearing routine. She did not return until July 6, having faithfully continued her non-weight

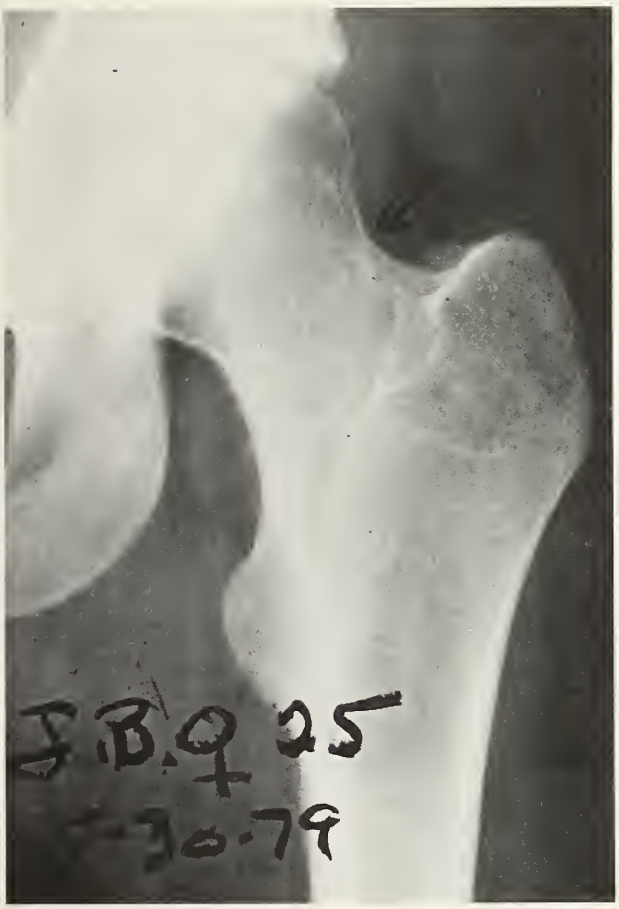


Figure 4. Arrow points to site of fracture of femoral neck which was not diagnosed at this time.



Figure 5. Arrow points to site of fracture. Note new bone formation on inferior surface of femoral neck.

bearing routine. X-rays at this time revealed a healing femoral neck fracture *(Figure 5)*. Weight-bearing was resumed but she was not permitted to run or jump. Swimming (no diving) provided an outlet for her energies and was permitted on an unlimited basis. Subsequent x-rays showed progressive and complete healing of the fracture. The patient has returned to full physical activity and is without symptoms.

Fortunately, both patients experienced solid bony union without residual disability, and both returned to normal activities. These cases illustrate the necessity for serious attention to aches and pains of these physically active individuals. If a fracture is not suspected and a patient is permitted to continue running, the fracture will probably displace, which then presents a difficult treatment problem with potential permanent disability.

Bone scanning techniques should aid in diagnosing these injuries or at least increasing the level of suspicion in spite of a normal x-ray. This would have been helpful in these cases. Scanning is also an aid in determining the state of bony union, the hot spot clearing as union progresses.

Conclusion

Joggers are usually healthy, hardy individuals, not inclined to complain, and they should be carefully examined. Good quality x-rays should be obtained and, if indicated, tomograms and bone scans should be utilized.

On the basis of the clinical history and a clinical suspicion of a fracture, the patient must be kept non-weight bearing. Swimming without diving is a good outlet for physical energies during the healing period.

Anxietas Tibiarum

Restless Legs and Headache

M. L. MASTERSON, M.D., *Troy*

Presented here is a review of the scanty literature on "restless legs," plus presentation of results of treatment with cyproheptadine HCl and serendipitous observation of relief of a particular form of headache. The results suggest a possible genetic factor involved in both conditions. The possibility of serotonin as a causative factor in both conditions is proposed.

Karl-Axel Ekbom in 1945 presented a description, analysis, classification, and suggested therapy for a previously poorly understood and largely ignored condition which he termed "restless legs."[1] Strictly defined, this disease (1) involved the lower extremities, most often below the knees; (2) occurred almost always at night, shortly after retiring; and (3) produced discomfort on a variable scale from severe pain down to an indefinable urge to move the legs, sufficiently strong to interfere with or prevent restful sleep. Relief could be obtained only by walking, often to the extent that sleep was possible only due to extreme fatigue rather than complete relief of the distress.

Ekbom had previously written short resumes and speculations on this condition, and reviewed the original article written by Wittmaack in 1861, as well as a few brief mentions of "anxietas tibiarum" consisting of both sensory and motor components. Later articles he reviewed consisted primarily of abstracts by various authors, describing the condition but making no attempt to explain a cause or propose a treatment. Many of the patients were termed "hysterical" and referred to psychiatrists for treatment.

In 1944, Allison's[2] more specific description outlined a condition he called "leg jitters." This was a "common minor ailment occurring when the legs get warm in bed and prevents one's going to sleep. An unlocalized restlessness is felt in one or both legs, not a pain but distinctly unpleasant, not relieved by voluntary or involuntary movements of the legs." Relief was obtained only by walking for ten minutes to an hour. Relief reportedly could be obtained immediately by chewing 0.01 gr nitroglycerin, suggesting a vascular origin. This abstract discusses nine other ailments, but this section consists only of two paragraphs, one on "leg jitters" and the other on treatment of nocturnal leg cramps with quinine. An editorial comment noted that "these discomforts are so common that such simple treatments would prove a boon to suffering humanity. A larger experience is certainly indicated."

Ekbom reported a series of 53 patients studied between 1943-1945 at the neurological outpatient services of the Serafimer Hospital (Serafimerlasarettet) in Stockholm. Thirty-four cases were moderately severe or severe, and the remainder had pain as a presenting complaint.

The single feature common to all cases was a crawling sensation deep in the tissues (usually in the calves), not relieved by massage or position change, requiring the patient to walk for varying periods to obtain relief. Some cases were mild; other cases were so severe that the patient contemplated suicide. Nearly two-thirds of the cases demonstrated coldness of the feet, and sometimes of the lower legs. Weakness of the legs was sometimes present. No objective findings could be elicited, the condition being entirely subjective. A definite familial pattern was elicited in many cases. Women predominated in the severe cases, but mild cases were about equally prevalent between the sexes.

Ekbom treated his cases with carbaminoylcholine (Doryl) and benzylimidazolin hydrochloride (Priscol) with improvement noted in 29 cases. Cases treated with placebos showed no improvement.

Since 1950, this author has experienced the "crawling" sensations in the calves as described by Ekbom. Onset invariably occurred shortly after retiring and before sound sleep began. No amount of position change afforded relief; this could be effected only by walking. The author's maternal grandmother experienced similar discomfort, which she termed "the walks." His mother, in younger years, spent many nights sitting and reading, constantly moving her feet and legs, maintaining that she "might just as well read, because I can't hold my legs still in bed anyway." The author's sister also

experienced similar discomfort for a few years but to a lesser degree.

In addition to the completely subjective crawling sensations in the calves, the grandmother, mother, and author also experienced unilateral, severe, and at times incapacitating headaches. There was no aura of any kind. Onset frequently occurred between midnight and 6 A.M., with pain sufficient to interrupt a sound sleep. Duration was from three or four to 36 hrs. The intensity of the pain was such that concentration, reasoning and reading were difficult, and nausea and occasional vomiting occurred. Unilateral photophobia and nasal congestion (on the side of the headache) were sometimes noted. The headaches occurred at intervals varying from once or twice/month to three times/week.

The author's youngest daughter, aged 29 yrs, recently described similar headaches, occurring at one- to two-week intervals, which she attributed to her menstrual cycle. These began two years ago.

For the author, this constellation of symptoms — present sometimes concurrently and sometimes separately (but always with the headache) — resulted in many diagnoses, including migraine, migraine variant, cluster headache, Horton headache, histamine headache, allergy, and hysteria. Many medications were prescribed, including salicylates, ergotrate maleate, Cafergot, Sansert, and opiates. Results were unpredictable and singularly unsatisfactory.

In February 1978, Prapas Gerjarusak, M.D., Leavenworth, obtained and shared with the author a copy of Ekbom's 1945 article. While the medications utilized by Ekbom were not totally effective, they held promise. The possibility that their action might have been that of serotonin suppression was considered, and another preparation, cyproheptadine HCl (Periactin, 4 mg) was chosen because it is a serotonin and histamine antagonist with anticholinergic and sedative effects. Cyproheptadine was determined to be appropriate because the author had taken antihistamines for many years, and anticholinergics during ulcer therapy, without relief of the leg discomfort.

Relief of the crawling sensation of restless legs occurred the first night cyproheptadine HCl was used. The crawling sensation has recurred since that time only on those occasions when cyproheptadine was not taken. Several weeks elapsed before it was noted that no further headaches had occurred. Since April 1978, Periactin, 4 mg, has been taken at 2 P.M., 6 P.M., and 10 P.M.; there have been no headaches, and restless legs have occurred only on those nights when Periactin was not taken.

Since May 1978, cyproheptadine has been prescribed for 15 patients, five of whom had complaints solely of restless legs, two with combined restless legs and unilateral headache of the type described, and eight with headache only.

All of those with characteristic restless leg symptoms experienced relief no later than the third night of medication, with no recurrence unless medication was missed. Seven of those with combined restless leg and headache symptoms experienced complete relief, and one discontinued medication on the third day because of the sedative effects. Seven of those with headache have had no further headaches (including one who experienced no headache during two weeks of cyproheptadine medication, experienced headache three days after discontinuing medication, and has had no further headaches during 3½ months of subsequent medication), and one has had partial relief, although dosage compliance is questionable in this case. Of those patients treated, all but four were female.

A familial relationship has been established in the following cases:

1. One male (the author) has an established history of the described headaches in four successive generations.
2. One male (the author) has an established history of restless legs in three successive generations.
3. One female reports a brother, sister, and daughter with the headaches.
4. One female reports a grandfather with the headaches.
5. One female whose restless legs ceased troubling her had "migraine" until about age 50 years, and her father had restless legs.
6. One female with the headaches reports that her mother experienced "migraine."
7. One female with the headaches has a son with "migraine."

Summary

Little material is available concerning "restless legs." A series of patients with restless legs, unilateral incapacitating headache, or a combination of the two were treated with cyproheptadine HCl (Periactin, 4 mg), a serotonin inhibitor. The high success rate in this small series suggests serotonin as a causative factor in both conditions, and that its suppression might provide relief for many patients presenting with these complaints. A possible genetic factor is suggested for one or both of these conditions.

(Continued on page 600)

A Six-Year Report

Spinal Deformity Screening in Kansas School Children

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Screening of Kansas public school children for spinal deformity has been in progress for six years, and 67 per cent of the school children are being screened. On the basis of this six-year experience, the basic principles of the program appear sound.

A DISEASE screening program, to be worthwhile, requires a reasonable understanding of the disease process, successful treatment made more effective by early detection, and a favorable cost benefit ratio. For spinal deformities, these prerequisites have largely been realized during the past 35 years. They include better definition of the idiopathic category following the virtual elimination of polio by the Salk and Sabin vaccines; population studies in Delaware,[1, 2] Los Angeles,[3] and Montreal;[4] natural history studies from Iowa[5] and Sweden;[6, 7] and introduction and perfection of the Milwaukee orthosis[8, 9] and Harrington instrumentation.[10]

Stimulated by these developments, several states and municipalities have initiated spinal deformity screening programs. In Kansas, we have tended to follow the pattern set by Minnesota.[11] Our voluntary public school screening program was initiated in 1974, as described in the *Journal of the Kansas Medical Society* in 1975[12] and supported by the Kansas Medical Society by resolution in 1976.[13] Reported here are the results of this six-year experience.

Methods and Materials

An extensive educational effort was launched in the form of lectures, school workshops, clinics, exhibits, and handouts. More than one thousand hours of educational time has been invested by the authors, district nurses, and concerned orthopedists within the state (Drs. Philip Baker, Earl Carlson, H. O. Marsh, Spencer McRae, Ernest Neighbor, and Carl Zacharias). In addition, presentations have been made to the Kansas Physical Therapy Association, Kansas Chapter of the American Academy of Pediatrics, the Kansas Radiological Society, the Kansas Orthopedic Society, and the Kansas Medical Society. To date, lay education has been less comprehensive.

The screening was performed by school nurses, county public health nurses, and in a few instances, other health care personnel — primarily physical therapists. Spearheading and overseeing the detection effort was the office of the State School Nurse Consultant. Following screening, diagnosis for those cases referred was provided by the primary care physician, and treatment — when necessary — by orthopedists.

Those targeted for annual screening were students in the public school system aged 10-13 yrs (grades five through eight). In Kansas, 93.7 per cent of school children are enrolled in public schools.

The program involves approval by the school administration and parent notification with the option for refusal provided. Screening is performed on the school premises using the locker room, gymnasium, nurses office, or modified classrooms. The sexes are segregated and privacy provided. Students are advised to wear appropriate clothing to include gym shorts, swimsuits, or shorts and halter tops. They are observed in the posterior upright, posterior forward bend, lateral upright, and lateral forward bend positions. Examination requires about 30 seconds per student. Physical education instructors or others can be helpful with the logistics of screening. The screener has three options: (1) normal (no spine deformity detected); (2) questionable — rescreen in three to six months; and (3) referral. Parents of those referred are provided notification forms which inform the physician of the findings and provide follow-up data for the screener.

Diagnosis is provided by the primary care physician who performs a confirmation examination and x-ray if, in his clinical judgment, it is indicated. Actions open to the primary care physician are discharge, if the curve detected is less than 10 degrees;

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TABLE I
NUMBER OF KANSAS SCHOOL CHILDREN SCREENED

Year	*CPHN**	*SN†*	*Total‡*	
1974-75	—	11,924	11,924	(10%)
1975-76	5,863	29,383	35,246	(28%)
1976-77	14,191	69,536	83,727	(67%)
1977-78	14,541	66,442	80,938	(65%)
1978-79	15,261	68,115	83,374	(67%)
1979-80	18,866	64,959	83,825	(67%)

* County Public Health Nurses.
† School Nurses.
‡ Based on 124,895 children in Kansas Public Schools, Grade 5-8 (Ages 10-13, 9-15-78).

follow at approximately four-month intervals if a curve is detected and is between 10 and 15-20 degrees; and referral to the orthopedist if a curve greater than 15-20 degrees is detected. In addition, if there are neurological changes on physical examination or congenital findings on spine x-rays, a referral is recommended.

Treatment is provided by orthopedists and consists of either a spinal deformity orthosis or surgical improvement and fusion.

Results

The number of Kansas public school children screened by year is shown in *Table I*. Most of the screening was performed by school nurses. During the first three years of the program, there was a progressive increase in the percentage of target-aged school children screened. However, this has been stable during the past three years with 65-67 per cent of the target-aged children being screened.

TABLE II
NUMBER OF SCREENED CHILDREN REFERRED

Year	*CPHN*	*SN*	*Total**	
1974-75	—	456	456	(3.8%)
1975-76	157	1,227	1,384	(3.9%)
1976-77	445	1,849	2,294	(2.7%)
1977-78	872	2,044	2,916	(3.6%)
1978-79	542	2,316	2,858	(3.4%)
1979-80	181	2,004	2,185	(2.6%)

* Per cent calculated from number of children screened.

During the 1978-79 school year, two-thirds of the 307 school districts in Kansas were fully or partially participating in a screening program.

The number of screened children referred by year is shown in *Table II*. The overall referral rate has varied between 2.6 and 3.9 per cent. The sex distribution of those referred has been assessed and is 1.5 girls : 1.0 boys.

The percentage of referrals completed and recommendations on completed referrals for the last four years is shown in *Table III*. The number of completed referrals has increased from 16 to 53 per cent.

The estimated incidence of treatment for the past four years is shown in *Table IV*. Calculation is based on the assumption that the completed referral group is representative of the total referral group. It appears that during the past three years the incidence of those being actively treated has been decreasing both for the braced group and for the surgical group.

Discussion

Our data do not provide exact epidemiological information. However, these results are comparable to the findings of other studies. In Minnesota, 75 per

TABLE III
COMPLETED REFERRALS AND MANAGEMENT RECOMMENDATION

Year	*Completed Referrals*		*Recommendations on Completed Referrals*			
	#	*%**	*None*	*Periodic Observation (& other)*	*Orthosis*	*Surgery*
1976-77	376	16%	34†	161	40	
1977-78	715	25%	169	477	57	12
1978-79	1,025	36%	288	649	76	12
1979-80	1,262	53%	267	905	76	14

* Per cent based on total number of students referred.
† Plus unknown treatment recommendation for 131.

TABLE IV
ESTIMATED TREATMENT INCIDENCE*

Year	*Orthosis*	*Surgery*	*Combined*
1976-77	—	—	2.9/1,000
1977-78	2.9/1,000	0.6/1,000	3.5/1,000
1978-79	2.6/1,000	0.4/1,000	3.0/1,000
1979-80	1.6/1,000	0.3/1,000	1.9/1,000

* Estimated Treatment Incidence =

$$\frac{\text{Number Referred} \times \% \text{ Completed Referrals Treated}}{\text{Number Screened}}$$

cent of all school children are being screened on a voluntary basis and approximately 3 per cent are being referred.[14] Numerous reports have documented that for small curves the female : male ratio is approximately 1.5 : 1.0.[3, 4] Treatment incidence has generally been reported to be about three/thousand (0.3%).[4]

Effectiveness of early detection by visual screening in lowering the incidence of treatment has recently been documented. During the ten-year period, from 1968-1978, surgical treatment for idiopathic scoliosis fell from 0.7/thousand to 0.4/thousand in the state of Gothenburg, Sweden.[15] During the past three year period we have shown a decrease in surgical treatment from 0.6 to 0.3/thousand. Surgical criteria have not changed during this time.

In Minnesota, not only has surgical treatment decreased, but treatment by spinal orthosis has likewise decreased.[14] This is due to the recent observation that not all idiopathic scoliotic curves of 20-30 degrees in the growing child will progress.[4] Although of shorter duration, our studies demonstrate the same trend. Thus early detection makes possible a decrease in the treatment incidence by allowing some patients to avoid surgery by early bracing, and for some with intermediate curves to avoid bracing by proving curve stability on longitudinal follow-up.

This raises the question as to what the incidence of patients requiring treatment really is. At the beginning of their study in 1967, Torell *et al.*[15] reported an incidence of curves greater than 40 degrees at 1.5/thousand and curves greater than 20 degrees at 3.2/thousand. Considering that stable curves of less than 40 degrees in the adult are not likely to cause difficulties, it could be estimated that between 1.5 and 3.2/thousand patients require treatment.

From the cost standpoint, early detection also appears to be effective. It has been estimated that for the growing adolescent, surgical treatment of idiopathic scoliosis is twice as expensive as orthotic treatment.[16] In Sweden, early detection has resulted from increasing public and professional awareness.[15] In the United States, the approach has been to develop school screening programs. In Minnesota it is estimated that the true cost/student screened is .29 cents and the time cost 35 cents.[14] In Kansas *no* additional health care personnel positions have been added for school screening. The program has been accomplished entirely within the framework of the existing health care delivery system. Based on an average school nurse salary of $7.50/hr, we estimate the cost of screening at less than 50 cents/student.

Thus, the desirability and effectiveness of early detection of spinal deformity seems well established. However, there are still questions, some of which can be currently answered and some of which cannot.

There is considerable debate about the recommended age of screening. Spinal deformities are most likely to develop or progress during the parapubescent growth spurt. For girls this begins at about age 10 yrs and peaks at about age 12 yrs 3 mos, which is about three months prior to menarche. For males the start and peak are about two years later.[17] It thus appears that the most reasonable age recommendation for screening is 10-14 yrs.

In an attempt to increase the percentage of vulnerable age children screened, nine states (*i.e.* Connecticut, Delaware, Florida, Massachusetts, North Carolina, South Carolina, New York, New Jersey, and Washington) have now mandated screening. For several reasons we are not convinced that mandation is desirable. While important, spinal deformity is not infectious and lacks the potential of epidemic spread. The number of students requiring active treatment has now been well documented at three or fewer/thousand. The natural history of adolescent kyphosis is largely unknown, making screening for that deformity (which is thought to occur at a slightly older age) questionable. Finally, mandation almost certainly results in the creation of a costly bureaucracy. We believe that a better method to increase the percentage of vulnerable age children screened is through education at all professional levels, along with dissemination of accurate and pertinent information to parents and children.

Another problem relates to the percentage of school children referred. In Kansas and in Minnesota[14] this has been about 3 per cent. This does not seem unreasonable considering that the incidence of curves greater than 6 degrees is between 4.5 and 13.6 per cent,[3] and the incidence of curves

greater than 10 degrees is 2 per cent.[4] However, it is important to keep in mind that the incidence of curve greater than 20 degrees is only 0.32 per cent,[15] and not all of these require treatment. Therefore, it should be possible for experienced nurses to keep their referral rate at approximately 2-3 per cent. For those children with minor trunk asymmetry and/or with small thoracic humps of less than half of a centimeter, the school nurse can make liberal use of a rescreen at three-to-six-month intervals. Students previously referred should also be included in this rescreen group. When referring a student to the primary care physician, it is desirable for the school nurse to make personal contact with the parents.

A question may arise concerning x-ray and the primary care physician's responsibility. In a well screened population, x-ray will usually be necessary. However, the primary care physician should use his judgment as to whether or not a standing spinal AP x-ray is indicated. If he is confident in his own knowledge and judgment, he should not feel that a routine x-ray is necessary, just because a student has been referred.

Follow-up of students with minor curves not requiring treatment can present a problem. For those students who seem to have a low likelihood of progression we have instructed the parents in the forward bending examination and ask that they check their child every three months and call for an appointment if they suspect progression.

Our goals for the future are to increase the percentage of children screened, increase the percentage of completed referrals returned for data, to continue the educational program, and to expand it to include parents and students. As a result, the desirable goals of early referral of significant curves and decreased incidence of unnecessary referrals should be realized. A constant effort is being made to improve our ability to predict curve behavior and to improve nonoperative results, including compliance with bracing. An effort is being made to stay abreast of research in this area in order to accommodate alternate legitimate treatment programs as they become available.

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Vox Dox

Vox Dox Editor:

"Wogging" — walking for pleasure and physical fitness — is a combination term derived from "walking" and "jogging." It is excellent exercise for virtually everyone, but especially for those who, for various reasons, do not run.

I am writing a book on this topic and would appreciate being advised of published articles on walking for health and pleasure. Those knowing of such information are asked to send it directly to me at the address below.

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Help When Needed

Lay Public Confident of CPR Skills

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SUDDEN DEATH from heart attack is the most significant medical emergency in America today. Approximately 1 million persons experience acute myocardial infarction annually and nearly 650,000 die. About 350,000 of these persons die before reaching the hospital, usually within two hours after the first symptoms occur. For many of these individuals, no evidence of coronary thrombosis can be found at autopsy, and coronary spasm is implicated as the cause of death. If appropriate treatment could be instituted promptly; many of these patients could be saved.[1]

Cardiopulmonary resuscitation (CPR) is an organized, effective system of emergency cardiac care. The steps involved in CPR are easily remembered as the ABCs: A — airway, B — breathing, and C — cardiac compression.[2] The first priority is to establish an airway and the second to initiate breathing by mouth-to-mouth technique. After a patent airway has been established and oxygen exchange has occurred in the lungs, the rescuer should assess the carotid pulse. If absent or questionable, he must begin chest compressions at once over the lower one-half of the sternum. Because cerebral anoxia and irreversible brain damage may occur within four to six minutes of cardiopulmonary arrest, it is urgent that basic life support be initiated promptly.

Since the adoption of these techniques of basic life support in 1956 by hospital cardiac arrest teams and mobile paramedic units, thousands of lives have been saved and restored to productivity.[3]

In recent years the American Heart Association has instituted programs to train the lay public as well as health professionals in rescue procedures. One of the long-range goals of the Kansas Heart Association is to have a trained rescuer at the side of every victim, and in survival action within one minute of sudden cardiac arrest. This philosophy implies that all citizens should avail themselves of the opportunity to learn CPR and join the lifesaving team. During the past year in the Wichita area, more than 4,000 health professionals and lay persons were trained and certified as CPR rescuers.

The American Heart Association proposes to train every man, woman, and child in the United States to perform cardiopulmonary resuscitation. Is this a realistic goal for the lay public? Will trained rescuers with no prior medical background actually use the technique in a crisis? A study in the Middle West indicates the affirmative.

The assumption is made that all persons who are trained and able to perform CPR would actually do so. This study was undertaken to determine the validity of that assumption.

A telephone survey was conducted among recently-certified CPR rescuers in the greater Wichita area to determine their feelings about the effectiveness of the course. By random selection, 131 persons completed the questionnaire. Seventy-nine (60%) were health professionals or were persons who had some paramedical training prior to taking the CPR course, which they felt prepared them for learning the technique. Fifty-two (40%) were lay persons with no prior paramedical instruction. Forty-seven (36%) were male and 84 (64%) were female. The majority — 81 persons (61.8%) — were between the ages of 18 and 40 yrs. Forty-eight (36.6%) were between 41 and 64 yrs. Two persons (1.5%) were aged 65 yrs. or above.

Method

Three groups of persons were chosen from rosters in the Wichita area from among those attending CPR classes during a twelve-month period. Individuals taking the course in October 1978, January 1979, and April 1979 were selected. All telephone calls were completed during the month of June 1979.

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A sample of at least 100 subjects was arbitrarily determined adequate for purposes of this study. Sixty-seven subjects were randomly chosen from each of the three groups to assure an adequate number of responses. Every third person was called in each group by random selection, and a reasonable attempt was made to contact each subject.

The Questionnaire

The following telephone questionnaire was developed by the authors for use in this study. Each question was to be answered *Yes, No,* or *Undecided.*

1. Do you consider your CPR training a worthwhile experience?
2. Do you think your CPR training was adequate; that is, do you feel that you would have a good chance of carrying it out successfully?
3. If you found yourself in a situation in which you were suddenly alone with a person who was not breathing and had no pulse, would you attempt to initiate CPR if this person were: your spouse? your child? your grandparent? a close friend? a casual acquaintance? a stranger? Would it make any difference in your response(s) if another person were present whom you knew also had CPR training?
4. If you were in a group or crowd and someone collapsed, would you assess the situation and take charge, rather than wait for someone else to do it?
5. Prior to taking the CPR course, did you have any formal schooling or experience which you feel helped prepare you for learning CPR?
6. Do you feel motivated to be recertified in one year?

Responses and Results

To question #1, 130 people replied yes; one no. The negative respondent had recently witnessed a drowning accident in which CPR rescuers had failed to revive a small boy.

On question #2, 113 replied yes, five no, and 13 undecided. Of those who were unable to answer yes, all but one were lay persons.

One hundred seventeen persons replied yes to all categories of question #3. Four subjects replied no or undecided concerning CPR on a spouse, two were unsure about attempting the technique on their own children, and two were undecided about attempting CPR on a grandparent. Two were undecided about initiating CPR on a close friend. Seven were negative or undecided about trying the technique on a stranger, citing "legal complications" as a possible restraining influence, along with the possibility of disease or poor hygiene in strangers. Only two health professionals were among those with negative or undecided responses to any part of this question. Both were nurses and both had doubts about performing CPR on a stranger. All 18 persons who were unable to say yes to all parts of the question replied that it would make a difference if an additional CPR rescuer were present.

To question #4, 80 persons replied that they would assume the responsibility in a respiratory or cardiac emergency, even though other people were present. Fifty-two of these were lay persons, 31 of whom had no prior medical instruction.

The final question had 121 affirmative responses. Four were negative (one a health professional) and six undecided (two were health professionals).

Following completion of the questionnaire, subjects were given an opportunity to make comments. The most frequently-made suggestion was that review and recertification intervals be shortened from one year to four or six months.

Conclusions and Recommendations

The primary motivation for this study was to discover whether lay persons with no prior medical knowledge actually feel they would use their CPR training in an emergency and whether they would be confident in doing so. The study seems to show that a majority would make the effort and think they have enough information and skill to be successful.

Several respondents suggested that for CPR to be truly learned, it must be "second nature" — or something a person could do automatically without having to think about it. A session or two only once a year may not be adequate to achieve this goal, particularly for the lay public. Perhaps the CPR committee should consider establishing some area reviewing centers so that people could review the materials and practice their technique at intervals of 12-16 weeks, between the annual recertification sessions.

The response to this study indicates that many people in the Wichita area are interested in CPR and appreciate the opportunity to learn it. As one subject put it, "The more people who know CPR, the better off we all are!"

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Physician Extender

The Army Physician's Assistant Revisited

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A NEW CATEGORY of health care personnel, known as the "physician's assistant" (PA), began training in early 1972 at the United States Army Medical Field Service School. This type of allied health personnel was to be an assistant to the physician, trained to do many tasks usually performed by a physician, but requiring less education. Although there had been much interest generated in this concept, and the Army was already committed to using these individuals to replace the battalion surgeon in the provision of primary health care to troops at the battalion aid station level, there had been some question about exactly what tasks these physician's assistants should be trained to do, and how such a person would function, especially in fixed medical facilities, both outpatient and inpatient, in the continental United States and elsewhere.[1]

Of vital importance was the attitude of the supervising physician. If Army physicians were generally opposed to allowing nonphysician personnel to perform some traditional physician tasks, or if they perceived a definite need for special training to manage such assistants, then there were obvious implications for educating and training of Army physicians, as well as for the training of PAs.

Another issue was the degree of independence appropriate for PAs. The medical literature recognized a need to provide primary health care to medically deprived areas of the United States; yet PAs — a possible solution to this problem — were generally required to work under the direct supervision of a physician, not geographically separated from him. As Carlson and Athelstan remarked, "If these assistants are distributed according to the same patterns as physicians in general, gaps in service to the poor and to residents of medically deprived sections of the inner city and rural areas will remain unfulfilled."[2]

It was suggested that Army physicians, with their experience with skilled corpsmen in such roles, would be the group of physicians most likely to approve of considerable independence for assistants practicing in remote locations. This hypothesis needed testing to demonstrate to civilian physicians that some members of the medical profession consider this use of PAs to be a reasonable solution to one of the nation's most serious problems — the maldistribution of physicians. This solution would, of course, also help to reduce the number of physicians required by the Army. It was deemed essential to determine if Army physicians generally conceived of this as a realistic, professionally acceptable solution; the problem of physician retention would be compounded if professionally unacceptable health care delivery systems were adopted by the Army Medical Department.

Little has been published concerning the training, utilization, and performance of Army physician's assistants. Indications are that the program has achieved outstanding success. Arrangements for required continuing medical education are critical for continued effectiveness.

In May 1971, in an attempt to confront these issues, the Health Care Research Division of the United States Army Medical Field Service School conducted an attitude survey of Army physicians concerning the PA. Questionnaires were mailed to a stratified random sample consisting of 1,409 Army physicians; response rate was 60.1 per cent. The results of this survey were published in June 1974 in *Military Medicine*.

An overwhelming majority (91.5%) of those responding felt that there was a need for PAs. More than 85 per cent of respondents indicated that they would request PAs to be assigned to them if they were working in an area where there was a physician shortage, and approximately half stated that they would request PAs even if there were no physician shortage. More than half expressed willingness to delegate to PAs 23 of 32 selected tasks in caring for all Army Medical Department beneficiaries in any practice setting. Most felt that they could manage at least two PAs and would be willing to share management of PAs with their colleagues. Approximately three-fourths of these physicians would allow the PA

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to handle most of the routine or minor problems, and more than 80 per cent believed that the quality of care would be improved or at least unchanged by the utilization of PAs. A majority of responding physicians approved of the assignment of PAs to remote locations with a fairly independent role in the care of active duty patients, but not of dependents.[3]

Faced with the end of the "doctor draft," three of the military services — Army, Navy, and Air Force — developed extensive educational programs to train large numbers of PAs. Their PA programs were designed to fill the void created by a lack of general practitioners.[4] The following is a review of developments since 1972 in terms of training, utilization, and performance of PAs in the United States Army.

In 1972 the Army Medical Department initiated a PA program which admitted candidates on a competitive basis after an intensive screening process of testing and personal interviews. Prerequisites for admission were: (1) a high school diploma or equivalent; (2) competence in the English language; (3) general technical score of 110 or higher; (4) any medical military occupational specialty, including three years of clinical experience; (5) United States citizenship; (6) recommendation from current commanding officer; (7) recommendation from a Medical Corps officer; (8) physical qualifications for warrant officer appointment; and (9) good moral character.[1]

The successful candidate undergoes two years of intensive medical training. The primarily didactic first year courses are conducted at Fort Sam Houston (Texas) by the Academy of Health Sciences and Baylor University. The second year is spent in a clinical practicum conducted at selected Army hospitals. This practicum enables the candidate to apply the theories learned in a medical facility supervised by physicians. After completion of the practicum, the PA candidate must take final examinations prior to certification. Finally s/he is appointed a WO1 or CW2 (depending on entry grade into the program) and awarded an Associate of Science diploma from Baylor University.[1, 5]

The Army is specific with respect to utilization of PAs; Army doctrine states that the PA "functions in the combat medical platoon under the direction of the medical platoon or medical section leader and receives technical supervision from the staff or command (brigade) surgeon."[5] The PA is responsible for the following:

1. Conducting and/or supervising training of battalion personnel in first aid, military sanitation, personal hygiene, medical evacuation procedures, and medical aspects of injury prevention;

2. Arranging for the conduct of the battalion preventive psychiatry program, with technical assistance from the division psychiatrist, to include training of battalion troop leaders in methods of preventing psychiatric disorders, especially combat exhaustion;

3. Establishing and operating a medical aid station;

4. Treating, within his medical ability, those patients reporting on sick call. Patients who require additional treatment or treatment beyond the medical ability or capability of the PA will be referred to the supporting medical company; and

5. Providing resuscitative care to wounded and injured to include

— establishment and maintenance of a patient's airway
— control of bleeding
— prevention and treatment of shock
— protection of wounds
— immobilization of fractures
— relief of pain and undue suffering.[5]

Further, the Army envisioned that after spending a tour in the maneuver battalion, the PA might then staff either a troop medical clinic or a health clinic.[1]

An evaluation of the efficacy of the Army PA program was first conducted in 1975 by Robinson and Thompson[4] who surveyed both supervising physicians and Army line commanders in an attempt to assess the acceptability of graduate military PAs providing primary health care under the supervision of physicians. This study also examined the perceptions of PAs as to their concept of the career program, acceptance, and continuing education.

Supervising physicians were asked to evaluate PAs in diagnostic and treatment skill areas. Questionnaires were submitted to 285 supervising physicians. Response rate was 44 per cent, and results were as follows:

Question: If you are unable to do a physical examination of a patient yourself, would you trust the PA's physical examination?

Response: Sixty-six per cent would accept in all but the most complicated cases, 31 per cent would always accept, and only one physician indicated that he would not accept.

Question: Given adequate diagnostic information, does the PA determine logical diagnostic possibilities?

Response: Fifty-two per cent in cases where two or more diagnoses might usually be considered, 44 per cent even in cases of difficult diagnoses, and four per cent only in most straightforward cases.

Question: Given an established diagnosis, does the PA plan treatment?

Response: Thirty-one per cent for even complicated management problems; 68 per cent for most commonly seen problems; 1 per cent limited to only the simplest.

Question: Would you trust this PA to write prescriptions in a routine sick call situation?

Response: Ninety-eight per cent, yes; 2 per cent, no.

Supervising physicians were also asked their perceptions of the PA's ability to interact with the patient and other health care workers. Overall, the findings indicate that the PA was perceived to "fit very smoothly and increase job effectiveness" in the medical environment.

Robinson and Thompson[4] submitted questionnaires to 81 battalion commanders of Army PAs. Response rate was 40 per cent, and yielded the following results:

Confidence: Ninety per cent were highly confident and 10 per cent were moderately confident of the PA's ability to perform medical examination and treatment.

Personal satisfaction: Ninety-one per cent were highly satisfied and 9 per cent were moderately satisfied with treatment received.

Receptiveness of unit personnel: Ninety-six per cent were highly receptive, and 4 per cent were moderately receptive.

Receptiveness of dependents: Eighty per cent were highly receptive, 17 per cent were moderately receptive, and 3 per cent were unreceptive.

Training of battalion personnel: Ninety-six per cent indicated the PA was used to train other medical personnel.

Battalion commanders uniformly expressed a high degree of acceptance and confidence in the program with no derogatory remarks.

Robinson and Thompson[4] survey questionnaires were submitted to 285 graduate PAs, and 64 per cent responded. The results indicated that 88 per cent of PAs were satisfied with their duties. Eighty-four per cent estimated that more than three-fourths of each duty day was devoted to primary patient care.

The PAs perceived the program to have been exceptionally well received by patients and by other health care personnel. Of those responding, 94 per cent felt that patient acceptance was extremely good, 6 per cent moderately good, and none felt that patient acceptance was poor. With regard to physician acceptance, 82 per cent felt that most physicians favor the concept, 15 per cent indicated moderate acceptance, and 1 per cent gave a neutral response.

The PAs voiced several concerns. A major concern is the rank structure. Eighty-eight per cent of respondents felt that their rank is not commensurate with required duty.

Another concern relates to continuing medical education. Since such education has become mandatory for recertification by the National Board of Medical Examiners, planning for it has become crucial. Robinson and Thompson point out that failure to provide such a program may have an impact on military retention.

In a more recently published study, Fitterer and Cochrane[6] attempted to evaluate the efficiency of physician's assistants during a period of two years in the U.S. Army 4th Division at Fort Carson, Colorado. They examined a program developed to utilize PAs in ambulatory patient care in troop medical clinics. They assessed the attitudes toward PAs of the physicians in the Medical Department Activity, the patients seen in the troop medical clinics, and the personnel working in the clinics.

The results indicated a high level of acceptance of PAs by all groups surveyed. The PAs were able to provide high quality medical care to Division personnel, and Fitterer and Cochrane felt that they had more rapport with patients and were more satisfied with their role than were their predecessors, the General Medical Officers. They felt that this program was also stimulating for the General Medical Officers who supervised the PAs.

Fitterer and Cochrane concluded that "the concept of the Physician's Assistant in the Army is no longer just a 'stop gap,' but is now truly the backbone of the ambulatory patient care in the Division." They further stated that the PA program "could help guarantee the future of Military Medicine." Like Robinson and Thompson, Fitterer and Cochrane warned that "Continued Medical Education is necessary to maintain proficiency of the PAs as well as to maintain their certification."

Little has been published concerning the training, utilization, and performance of Army physician's assistants. Nonetheless, based upon review of the limited published information concerning the Army PA program, it can be concluded that the program has thus far achieved outstanding success. Continuing education is crucial for insuring continued success. PAs will doubtless persist in their pressure for commissioned officer status.

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(Continued on page 579)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Office Spirometry

GERALD R. KERBY, M.D.,* *Kansas City, Kansas*

WHAT KINDS of testing capability, beyond the usual history and physical examination, are appropriate in the office of the primary care physician? Answers to this question are as varied as individual personalities. Attributes of a useful office test include simplicity, accuracy, reasonable cost, and the ability to provide useful data for decision making not provided by clinical examination or other available tests. The case for including simple pulmonary function testing in the office is addressed in this article.

Which Test(s)?

A wide variety of tests for various aspects of lung function has evolved from the physiology laboratory into the pulmonary function laboratory. The proliferation of electronics has resulted in the inclusion of too many of these in the average battery of tests. A pulmonary function report often resembles a bank statement resulting in confusion for the average clinician no longer able to recall which numbers are pertinent, and which merely interesting. For clinical purposes most of the useful information is provided by the forced vital capacity curve and analysis of arterial blood gas. Blood gas analysis requires expensive equipment and is best relegated to the hospital laboratory. The forced vital capacity curve is easily and quickly measured on simple, reliable, and relatively inexpensive equipment. The test can be accurately performed by any office personnel after brief training.

* Professor of Medicine, University of Kansas School of Medicine-Kansas City.

Address reprint requests to Dr. Kerby, UKSM-KC, 39th & Rainbow Blvd., Kansas City, KS 66103.

FIGURE 1

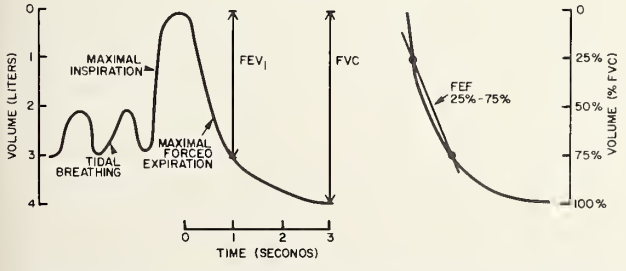


Figure 1. Representative tracing produced by office spirometer.

Which Number(s)?

Practical spirometers for office use produce tracings as illustrated in *Figure 1* with volume plotted on the ordinate and time on the abscissa. Some record the entire maneuver and others only the forced expiratory curve. The useful information is provided by three numbers:

Forced vital capacity (FVC): The total volume of air expelled from the lungs during maximal forced expiration of the vital capacity. This is directly measured from the volume calibration printed on the recording paper.

Forced expiration volume in one second (FEV_1): The volume of air expelled during the first second of the forced expiratory maneuver. This is measured by locating the point on the curve one second after initiation of the forced expiratory maneuver and reading the volume directly from the volume printed on the recording paper. In addition to the volume itself it is useful to express the ratio of FEV_1 to FVC:

$$\left(\frac{FEV_1}{FVC} \times 100 \text{ or } \frac{FEV_{1\%}}{FVC}\right).$$

Forced expiratory flow between 25 and 75 per cent of the forced vital capacity ($FEF_{25-75\%}$). The average rate of airflow during the middle half of the forced vital capacity. Measured by dividing the FVC by four and locating the points on the curve representing the first and last fourths. A line drawn between these two points is extended until it crosses two time lines one second apart. The volume between the two time lines is observed and represents the flow in liters/second.

The values obtained are compared with predicted normals derived from large population groups. Tables or nomograms of predicted normal values are usually provided with the spirometer. They are contained in the booklet *Chronic Obstructive Pulmonary Disease* which can be obtained without charge from the local office of the American Lung Association. The range of normal for FVC and FEV_1 is about ±20 per cent of predicted. For $FEF_{25-75\%}$ the range is ±40 per cent of predicted. The ratio $FEV_{1\%}$ should be greater than 75 per cent in young adults and greater than 70 per cent in older adults.

Interpretation

Abnormal spirometric values can be classified into three patterns: restrictive, obstructive, or both restrictive and obstructive *(Table I)*. The physiologic mechanisms and common pathologic causes of the abnormal patterns detected by spirometry are listed in *Table II*.

Indications for Spirometry

Periodic Health Examinations. Symptoms, physical examination, and chest x-ray are often insensitive in detecting lung disease in early stages, particularly obstructive lung diseases. Spirometry can detect these much earlier. The $FEF_{25-75\%}$ is abnormal in about half of chronic smokers. Recent studies indicate those smokers susceptible to the development of progressive airflow obstruction can be detected at age 40 years by the presence of an abnormal FEV_1.

TABLE I
PATTERNS OF SPIROMETRIC ABNORMALITIES

	Restrictive	*Obstructive*	*Both*
FVC	Low	Normal	Low
FEV_1	Low	Low	Low
$\frac{FEV_{1\%}}{FVC}$	Normal	Low	Low
$FEF_{25-75\%}$	Normal	Low	Low

TABLE II
USUAL CAUSES OF SPIROMETRIC ABNORMALITIES

Abnormal Pattern	*Physiologic Mechanism*	*Pathologic Cause(s)*
Restrictive	Decreased lung compliance (stiff lung)	Pulmonary edema Pulmonary fibrosis idiopathic silicosis asbestosis Pulmonary granuloma sarcoidosis
	Decreased thoracic compliance (stiff thorax)	Obesity Ankylosing spondylitis Kyphoscoliosis
	Neuromuscular (weak respiratory muscles)	Myasthenia gravis Muscular dystrophy Spinal cord injury Guillain-Barre
	Space occupying process	Pneumothorax Pleural effusion Ascites Intrathoracic tumor
Obstructive	Tracheobronchial obstruction	Chronic bronchitis Asthma Tracheal stenosis
	Increased lung compliance (low elastic recoil, floppy lung)	Emphysema Asthma
Both	Air trapping (large residual vol.)	Emphysema Asthma
	Multiple mechanisms (Both airflow obstruction and restrictive disease)	Various combinations e.g. asthma and obesity

Guide to Therapy. Some measure of lung function should be part of the routine follow up examination of patients being treated for lung disease. This is especially important in asthmatic patients who often fail to perceive significant changes in airflow obstruction. Detection of early deterioration or the response to bronchodilator or corticosteroid therapy requires some measure of lung function since symptomatic changes are often misleading.

Preoperative Evaluation. The incidence of postoperative pulmonary complications leading to increased morbidity and prolonged hospital stay is several times higher in patients with abnormal lung function. This incidence can be greatly reduced by utilizing preoperative pulmonary function testing to

TABLE III
RECOMMENDED OFFICE SPIROMETERS

Instrument	*Manufacturer*	*(Price July 1980)*
Survey Spirometer	W. E. Collins 220 Wood Road Braintree, MA 02184	$795.00
Breon Spirometer	Breon Laboratories 90 Park Avenue New York, NY 10016	$900.00
Vitalograph	Vitalograph Medical Instr. 8347 Quivira Road Lenexa, KS 66215	$998.00
Pulmonor II	Jones Medical Instr. 200 Windsor Drive Oak Brook, IL 60521	$1100.00

recognize risk followed by appropriate therapy. Such patients at risk are generally not detected by a preoperative chest x-ray.

Course and Prognosis of Disease. The clinical course and prognosis of most patients with lung disease is best defined by the degree of abnormal lung function. Serial documentation of lung function is useful in decisions regarding therapy; it is particularly useful when difficult decisions such as mechanical ventilation must be faced.

Disability and Compensation. Most decisions regarding disability and compensation in patients with lung disease require documentation of lung function.

Which Spirometer?

Desirable attributes for an office spirometer include accuracy, reliability, simple operation, and reasonable cost. Recommended spirometers measure lung volume with a bell or bellows that is directly linked to the recording device. This results in greater reliability and accuracy. Devices that measure air flow and electrically integrate flow to volume are not recommended. The flow sensors in these are inherently nonlinear and require expensive electronics for poper compensation. Calibration requires equipment not available in the average office. Recommended spirometers are listed in *Table III*.

Summary

The clinically most important tests of lung function can be measured with an office spirometer. Such tests provide information useful for patient management that is often not obtained from history, physical examination, or chest x-ray. Currently available instruments are reliable, easy to operate, and moderate in cost. They are recommended for inclusion in the office of all physicians seeing patients with potential or established lung disease.

Self Assessment Questions

1. Other than analysis of arterial blood gases a forced vital capacity curve is the most helpful test for assessment of pulmonary disease. (T or F)
2. In bronchial asthma changes in patients' symptoms are an insensitive measure of changes in lung function. (T or F)
3. The forced expiratory volume at one second will be low in obstructive lung disease and normal in restrictive lung disease. (T or F)
4. Emphysema or asthma may be associated with restrictive and obstructive lung disease. (T or F)
5. Pathologic causes of restrictive lung disease include decreased lung compliance, decreased thoracic compliance or weak respiratory muscles. (T or F)

(Answers on page 600)

Physician Extender

(Continued from page 576)

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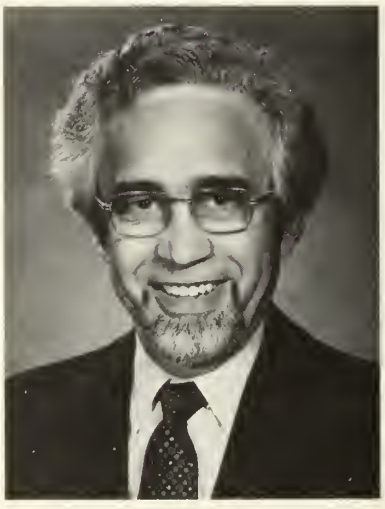
The President's Message

The beautiful autumn weather was nearly perfect, which made the football games all the more enjoyable for the spectators. We have seen some spectacular runs this year along with some thrilling pass plays. One can't help but reflect on differences of one school team from another, or one year's team from the next at the same school. Is it a new quarterback or a new running back?

The answer, of course, is that it is many factors. Passing the ball requires someone to catch it! A good passing quarterback is made great by skilled receivers who perform their jobs with perfection. And the "long bomb" takes backs who can block to give the passer time for the play to develop and a line to keep the defensive line contained so the quarterback can see his target.

The running back needs speed and dexterity, but it is the key block in front of him that often accounts for the gained yardage. The kicker needs not only a strong leg, consistency and perhaps the wind at his back, but also time to get the kick off . . . and that's provided by the other 91 per cent of the team in front of him.

So it is with the Medical Society. Our success comes from the various component societies throughout the state, the many committees and commissions of our state Society which represent literally thousands of man hours per year in dedicated volunteer work, our Councilors and delegates who set the policies of our Society after extensive deliberations, and the office staff who maintains the day-to-day functions of our Society.



I appreciate the opportunity of being your figurehead, but I fully understand that any success we may experience as a Society depends on *you*. Thanks for your efforts; they are not going unappreciated.

The other officers and staff of The Kansas Medical Society join me in wishing each of you happy holidays, and joy and good health in 1981.

President



Editorial COMMENT

In Case of Overdose

Those who pursue the ancient and noble art of commentary know no greater joy than to come upon a matter so speculative and far off in occurrence that no one knows what it will look like when it arrives. This permits observations so equivocal that accuracy can later be claimed regardless of the outcome.

This introduces the subject of the predicted glut of physicians on the medical market. As with the growing up of children, we are hardly prepared for the suddenness of change from the shortage of yesterday to the oversupply we'll have tomorrow. Although authorities are about evenly divided between those who denied there was a shortage in the first place and those who deny there will be an excess as claimed, we can say right off that whoever could accomplish such a quick turn-around should have been the one to be elected president.

While our interpretations don't have the weight of the scientific analysis that produced the prodigious estimate, our doubt that the event will develop in a straightforward form stems from the complexities of physician distribution and utilization, medical school admissions policies and methods, the irresistible social forces and the immovable bureaucratic objects — those same factors we contended with in the lean years in whatever form will be popular in that time.

The lay press has examined the matter with varying conclusions but predictably critical attitudes toward physicians. A physician excess will result in higher fees, they foresee, since physicians will try to maintain their incomes in the face of reduced patient loads. (On the other hand, they were certain that the physician shortage was engineered by physicians in order to limit the supply and command exorbitant fees.) Once again, we are confronted with the public's (or its mouthpieces') refusal to understand that physician service is not a commodity subject to the traditional laws of supply and demand.

The supply of physicians will, of course, increase — that was the purpose of the rather strenuous effort of recent years — and there are numerous channels for carrying off this spate. For one thing, the cause of non-illness which is approaching a state of sanctity, couldn't have asked for a bigger boost than an oversupply of physicians. Since the oldsters are likely to keep a firm grip on the sick, the new arrivals may decide that health is what they wanted to practice all along. Various plans to turn health into a viable segment of medical service are off and running — often on top of each other. This source of person-power (ugh), an available cadre of physicians, may change the temper of the profession's attitude toward the concept in a medical generation or two.

On a slightly different tangent, there is the matter of physician extenders who have appeared on the scene in recent years. Their development has been multifactorial — the need for a level of medical service of less than full physician expertise or attendance, the rapport these people can often achieve with patients not only by force of their capabilities but because the physician's role may inhibit it, the physical and geographic limitations imposed on the physician of limited time and strength. Since physician's assistants, nurse practitioners, and various technologists have moved into this area, it may be interesting to see what happens if, in their excess, physicians decide they want to cover both sides of the street after all. At the least, the future of programs of training for such ancillary personnel would seem to be problematic.

In another direction, concern has been voiced in academic circles about the relatively small number of medical graduates electing research careers. This has caused some expression that the current crop lacks something in dedication or intellect but sooner or later has been related to the lower incomes of the academics. If the market place gets too crowded, the temple may prove to have a greater attraction. At first sight, these recruits may lack something in appeal for the established inhabitants as they will appear to be lacking the True Spirit and somewhat prostituted (if prostitution can be somewhat) by the economic coercion. The eternal struggle for founda-

tion and government grants which make up standard fare on the academic menu will probably have more to do with the reception of novices than the excess of candidates. Anyway, it has been suggested that we have been assaulted with too much new material and perhaps it's time for a moratorium in that area. But if the news reports circa 1990 tell of a trend toward research by new physicians, remember you saw it here first.

Then there is the prospect, probably a matter of wishful thinking, that the oversupply will solve the ubiquitous and vexing problem of physician distribution. This is based on the theory that if medical bodies are piled high enough, they will spread out and seek the lower levels that have been deprived. This is a tempting thought but any indication that such areas are receiving favorable attention should be interpreted as a quantitative effect only after discounting the financial enticements and sales promotions already in effect, not to mention a very real determination on the part of many young physicians to seek such service voluntarily.

This "shortage" condition, whatever it's character, has been bound up with another phenomenon of contentious effect, the influx of foreign medical graduates. There have already been signs of a slowing down in this area but there will remain the problem of that special variety of FMG, the American student who has gone outside for training and returns, hoping to practice at home. On the one hand, the return of restrictions on medical school enrollments to curb the tide of domestic graduates will increase the pressure for such extraterritorial efforts (as well as the diversion of prospective medical students into ancillary areas) but those graduates will surely find themselves at an increasing disadvantage as this surplus becomes real.

It has been suggested that we can get a preview of the physician surplus by looking at the situation in San Francisco which is reported to have a demonstrable excess — one physician for every 216 persons which, despite the arguments as to what constitutes a surplus, makes it hard to deny that San Francisco is so blessed. And younger physicians, as expected, are the most vulnerable to the effects. The validity of San Francisco's condition as a subject for formal statistical study is suspect, however, by the simplicity of its etiology: the situation has arisen for the simple reason that too many physicians like the place and want to live there. Many have moved in and many younger ones who have trained there are loath to leave, hence the foretaste of 1990. (Of course, any TV fan can tell you that given the crime in the streets and the way the motorists slalom over the hills, there should be no end of demand for emergency room physicians.)

Admitting the unscientific nature of the volume and character of the sampling and the anecdotal reporting, the problem seems to reflect a grim determination of the principals to adhere to their personal desires rather than any combination of deep social or professional elements (and don't overlook this imponderable but potent effect on any future scene). The immediate result is that these younger ones, trying to get a foothold, have to resort to moonlighting. A physician oversupply, it seems, requires one to run faster just to stay in place. Some have left the area — but just barely as they have tended to settle in the environs. So much for redistribution to deprived areas. So it should be recorded that this very human characteristic of personal choice immediately skews the observations, and our sociologic interpretations are already messed up. Well, at least we recognize it and that's more than the sociologists do.

So far in San Francisco, one of the things the surplus hasn't done is reduce fees. Those physicians seeking to develop practices are not doing so by offering bargain rates but by expending more energy. It is reported, however, that patients are receiving certain benefits. Physician availability is such that long waits for attention are less and less common. Moreover, with a lighter patient load, physicians are devoting more time to the individual patient — listening more and providing minor services that were previously lost in the pressure of time. Undoubtedly, this will shortly be reported by the press as an effort to run up the bill.

So our pebble glass crystal ball is trying to tell us something but we can't quite make it out. We have a hunch the message is that a lot of the changes that the social pressures have been promoting will move faster if and when there are too many of us not because of the intrinsic value of the changes as much as the simple human reactions to the condition. Many of the features of medical practice that have been thought to be essential and inviolate will give way.

Meantime, to the social planners there will never be enough physicians until they are falling over themselves to participate in the plans. To the third parties, there will never be enough until they welcome the fees and conditions offered. To the student, there will always be need for one more. To the academic, there are too many clinicians who don't know the score. To the clinicians, there are too many academics who don't know the score. To the patient,

(Continued on page 600)

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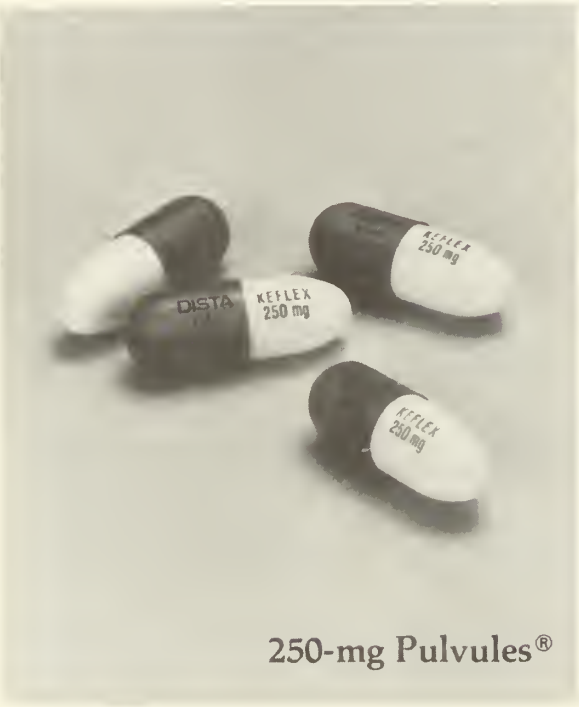
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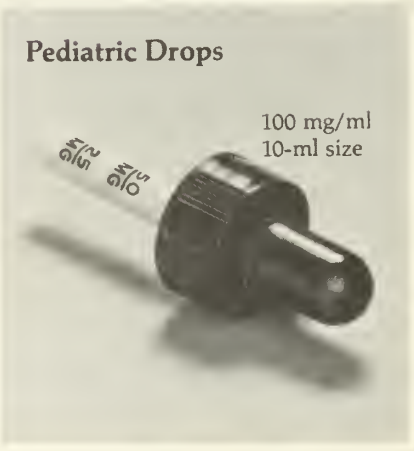
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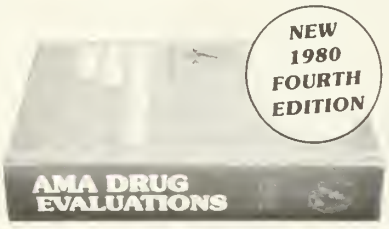
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Writing Your Congressman

HON. MORRIS K. UDALL*

Wednesday, November 2, 1977

Surprisingly few people ever write their Congressman. Perhaps 90 per cent of our citizens live and die without ever taking pen in hand and expressing a single opinion to the man or woman who represents them in Congress — a person whose vote may decide what price they will pay for the acts of Government, either in dollars or in human lives.

This reluctance to communicate results from the typical and understandable feelings that Congressmen have no time or inclination to read their mail, that a letter probably will not be answered or answered satisfactorily, that one letter will not make any difference anyway. Based on my own 16 years' experience, and speaking for myself, I can state flatly that most of the notions are wrong. On several occasions a single, thoughtful, factually persuasive letter did change my mind or cause me to initiate a review of a previous judgment. Nearly every day my faith is renewed by one or more informative and helpful letters giving me a better understanding of the thinking of my constituents.

Mail Is Important

Mail to today's Congressman is more important than ever before. In the days of Clay, Calhoun, Webster, and Lincoln, Congressmen lived among their people for perhaps nine months of the year. Through daily contacts in a constituency of less than 50,000 people — I represent ten times that many — they could feel rather completely informed on their constituents' beliefs and feelings. Today, with the staggering problems of Government and increasingly long sessions, I must not only vote on many more issues than early-day Congressmen but I rarely get to spend more than 60 days a year in Arizona. Thus my mailbag is my best "hot line" to the people back home.

Some Fundamentals

Here are some suggestions that apply to all congressional mail:

Address it properly: "Hon. _____, House Office Building, Washington, D. C. 20515." Or "Senator _____, Senate Office Building, Washington, D. C. 20510." This may seem fundamental, but I once received a letter addressed like this: "Mr. Morris K. Udall, U. S. Senator, Capital Building, Phoenix, Arizona. Dear Congressman Rhodes. . . ."

Identify the bill or issue: About 20,000 bills are introduced in each Congress; it's important to be specific. If you write about a bill, try to give the bill number or describe it by popular title ("clean air," "minimum wage," etc.).

The letter should be timely. Sometimes a bill is out of committee, or has passed the House, before a helpful letter arrives. Inform your Congressman while there is still time to take effective action.

Write the Right People

Concentrate on your own delegation. The representative of your district and the senators of your State cast your votes in the Congress and want to know your views. However, some writers will undertake to contact all 435 Members of the House and 100 Senators, who cast votes for other districts and other States. If you happen to be acquainted personally with a Member from Nebraska, he might answer your letter, but there is a "Congressional courtesy" procedure which provides that all letters written by residents of my district to other Congressmen will simply be referred to me for reply, and vice versa.

Be Brief

Be reasonably brief: Every working day the mailman leaves some 150 or more pieces of mail at my office. Tomorrow brings another batch. All of this mail must be answered while I am studying legislation, attending committee meetings and participating in debate on the House floor. I recognize that many issues are complex, but your opinions and arguments stand a better chance of being read if they are stated as concisely as the subject matter will permit. It is not necessary that letters be typed — only that they be legible — and the form, phraseology and grammar are completely unimportant.

In the course of my years in Congress I have received every kind of mail imaginable — the tragic, the touching, the rude, the crank; insulting, persua-

* U. S. Representative from the State of Arizona.

sive, entertaining, and all the rest. I enjoy receiving mail, and I look forward to it every morning; in fact my staff people call me a "mail grabber" because I interfere with the orderly mail-opening procedures they have established. Whatever form your letter takes I will welcome it. But to make it most helpful I would suggest these "dos" and "don'ts."

Do: Write Your Own Views

Write your own views — not someone else's. A personal letter is far better than a form letter or signature on a petition. Many people will sign a petition without reading it just to avoid offending the circulator. Form letters are readily recognizable — they usually arrive in batches — and usually register the sentiments of the person or lobbying group preparing the form. I regret to report that form letters often receive form replies. Anyway, I usually know what the major lobbying groups are saying, but I don't often know of your experiences and observations, or what the proposed bill will do to and for you. And I often am not fully aware of new conditions and developments in Arizona. A sincere, well-thought-out letter from you can help fill this gap.

Do: Give Your Reasons

Give your reasons for taking a stand. Statements like "Vote against H.R. 100; I'm bitterly opposed" don't help me much. But a letter which says "I'm a small hardware dealer, and H.R. 100 will put me out of business for the following reasons . . ." tells me a lot more. Maybe I did not know all the effects of the bill, and your letter will help me understand what it means to an important segment of my constituency.

Do: Be Constructive

Be constructive. If a bill deals with a problem you admit exists, but you believe the bill is the wrong approach, tell me what the right approach is.

If you have expert knowledge, share it with your Congressman. Of all the letters pouring into a Congressman's office every morning, perhaps one in a hundred comes from a constituent who is a real expert in that subject. The opinions expressed in the others are important, and will be heeded, but this one is a real gold mine for the conscientious Member. After all, in the next 9 to 10 months I will have to vote on farm bills, transportation bills, space, health, education, housing, and veterans' bills, and a host of others. I can't possibly be an expert in all these fields; many of my constituents are experts in some of them. I welcome their advice and counsel.

Do: Say "Well Done"

Say "well done" when it's deserved. Congressmen are human, too, and they appreciate an occasional "well done" from people who believe they have done the right thing. I know I do. But even if you think I went wrong on an issue, I would welcome a letter telling me you disagreed: it may help me on another issue later.

My list of "don'ts" would include these:

Don't: Threaten or Promise

Don't make threats or promises. Congressmen usually want to do the popular thing, but this is not their only motivation. Nearly all the Members I know want, most of all, to do what is best for the country. Occasionally a letter will conclude by saying, "If you vote for this monstrous bill, I'll do everything in my power to defeat you in the next election." A writer has the privilege of making such assertions, of course, but they rarely intimidate a conscientious Member, and they may generate an adverse reaction. He would rather know why you feel so strongly. The reasons may change his mind; the threat probably won't.

Don't: Call Names

Don't berate your Congressman. You can't hope to persuade him of your position by calling him names. If you disagree with him, give reasons for your disagreement. Try to keep the dialogue open.

Don't pretend to wield vast political influence: Write your Congressman as an individual — not as a self-appointed spokesman for your neighborhood, community, or industry. Unsupported claims to political influence will only cast doubt upon the views you express.

Don't: Become a "Pen Pal"

Do not become a constant "pen pal." In a newsletter appealing for more constituent mail I do not want to discourage letters, but quality, rather than quantity, is what counts. Write again and again if you feel like it, but do not try to instruct your Congressman on every issue that comes up. And do not nag him if his votes do not match your precise thinking every time. Remember, he has to consider all his constituents and all points of view. Also, keep in mind that one of the pet peeves on Capitol Hill is the "pen pal" who weighs the mail down every few days with long tomes on every conceivable subject.

Don't: Demand Commitments

Do not demand a commitment before the facts are in. If you have written a personal letter and stated

your reasons for a particular stand, you have a right to know my present thinking on the question. But writers who "demand to know how you will vote on H.R. 100" should bear certain legislative realities in mind:

On major bills there usually are two sides to be considered, and you may have heard only one.

The bill may be 100 pages long with 20 provisions in addition to the one you wrote about, and I may be forced to vote on the bill as a whole, weighing the good with the bad.

It makes little sense to adopt a firm and unyielding position before a single witness has been heard or study made of the bill in question.

A bill rarely becomes law in the same form as introduced. It is possible that the bill you write me about you would oppose when it reached the floor.

Remember: Bills Change

The complexities of the legislative process and the way in which bills change their shape in committee is revealed by a little story from my own experience. One time a few years ago I introduced a comprehensive bill dealing with a number of matters. I was proud of it, and I had great hopes for solving several perennial problems coming before Congress. However, after major confrontations in committee and numerous amendments I found myself voting against the "Udall bill."

Finally . . .

It is now November 1977, approaching the halfway point of the 95th Congress. Before 1978 is history the House clerk will record my votes on more than 450 issues. But in a very real sense these will not be"my" votes alone: they will be yours too. There are more than 500,000 Americans in the Second Congressional District of Arizona but when the clerk calls the roll he calls only my name. Thus these votes I cast will speak for you in the decisions our country must make in the next two years.

I need your help in casting these votes. The "ballot box" is not far away. It is painted blue and it reads "U. S. Mail."

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

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Obituaries

Richard F. Conard, M.D., 64, Emporia, UKSM 1942, died April 8, 1980.

George E. Cowles, M.D., 84, Wichita, UKSM 1921, died December 7, 1979.

Clarence A. Gripkey, M.D., 75, Kansas City, UKSM 1930, died April 1, 1980.

Chester W. Haines, M.D., 79, Haven, Northwestern 1928, died July 5, 1980.

Norman E. Hull, M.D., 56, Hays, UKSM 1952, died January 3, 1980.

Henry W. Kassel, M.D., 74, Lakewood, CO (formerly Kansas City), UKSM 1931, died February 28, 1979.

William J. Madden, M.D., 56, Lincoln, UKSM 1951, died May 13, 1980.

Donald N. Medearis, Sr., M.D., 79, Kansas City, Harvard 1927, died April 22, 1980.

James T. Moy, M.D., 66, Wichita, Jefferson Medical College 1940, died July 17, 1980.

A. S. Reece, M.D., 79, Gardner, UKSM 1929, died April 8, 1980.

Albert J. Rettenmaier, M.D., 84, Kansas City, Creighton 1926, died November 26, 1979.

Glen R. Shepherd, M.D., 60, Corona Del Mar, CA (formerly Kansas City), UKSM 1944, died September 1, 1979.

Eugene A. Walsh, M.D., 62, Onaga, Creighton 1942, died November 5, 1979.

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WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.
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DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.
HOW SUPPLIED

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Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
Alcohol	5%

Ru-Tuss Expectorant is an oral antitussive, antihistaminic, nasal decongestant and expectorant preparation.
INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold.
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PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease. Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.
ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.
DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.
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WHEN ANXIETY AND TENSION MAGNIFY PAIN

IN MUSCULOSKELETAL DISEASE*



A non-narcotic one-two punch against pain, with concurrent relief of anxiety/tension

EQUAGESIC®

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgement and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentrations in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphetamine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during continuous administration of meprobamate are reported; most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

Wyeth Laboratories
Philadelphia, Pa. 19101

™

FOR MODERATE PAIN

A therapeutic dose of acetaminophen in *one* tablet

A therapeutic dose of two complementary analgesics

The convenience and economy of a dosage schedule of one tablet, every four hours as needed



WHY NOT WYGESIC® ℠

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

WYGESIC—Abbrevieted Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE. Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently, physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY. Therefore, propoxyphene should not be used in pregnant women unless, in the judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS. The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antagonists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most ceses of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatel.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiretory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An eppropriate dose of one of these antagonists should be administered, preferably IV, simultaneously with efforts at respiretory resuscitation and the antegonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to e narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used beceuse of their fendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and or induction of emesis may be indicated. Activated charcoal is probably ineffective unless edministered almost immedietely after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since ecetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteamine) or other thol compounds may protect against liver demage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation es a less toxic alternative to mercaptamine, which may cause enorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information. (JAMA 237:2406-2407, 1977)

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Domestic distribution of the Dalkon Shield by A. H. Robins Company was discontinued in June 1974.

Recent medical literature tends to suggest a relationship between duration of use of inert IUDs and risk of pelvic infection, particularly when the causative organism is *Actinomyces israelii*.

As present users of the Dalkon Shield are in the long-term category, A. H. Robins now recommends removal from all patients, even those who are asymptomatic. It is also suggested that the removed IUD and all adherent material be submitted to a clinical pathology laboratory for examination and that the patient be appropriately followed and treated as clinically indicated.

For further information regarding the Dalkon Shield, see Horbelt, Douglas V.; Roberts, Daniel K. and Adamson, Harold W.: Studies in Translocation: Dalkon Shield — Insertion, Perforation, and Migration. *J. Kans. Med. Soc.* 80:323-326, 1979.

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Anxietas Tibiarum

(Continued from page 567)

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Office Spirometry

(Continued from page 579)

1. *True*
2. *True*
3. *False*
4. *True*
5. *True*

Suggested Readings

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In Case of Overdose

(Continued from page 582)

there are too few when he's hurting and too many when the bills come in. To the younger physicians, there are too many senile old codgers and to the older ones too many half-baked young ones.

Supply, it would seem, is in the eye of the beholder. — *D.E.G.*

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Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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